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Physiology and
Pathology of
INFANT NUTRITION

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Infant Nutrition

Completely Revised Second Edition

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Introduction

THE TOPICS of nutrition and nutritional disturbances in infancy seem no longer timely. Through improvement of living conditions of large groups of the population in many countries and through the development of social services and betterment of milk hygiene the problems of artificial infant feeding have found their solution. The constantly decreasing infant morbidity and mortality are the numerical results of this progress. The prevention and treatment of acute and chronic nutritional disturbances has led to the successful raising of artificially fed infants. The improvement of methods in treating acute diarrheal diseases which were developed in the past ten years and the possibility of successfully raising prematurely born infants contributed considerably to this decrease in infant mortality.

This desirable state of affairs has as yet not been attained in all the countries of the world. Economic, cultural and social conditions still hamper development in some places. War, famine, mass migration and concentration of large groups of the population in camps have shown how easily and rapidly seemingly established standards can again be destroyed. Unfavorable hygienic conditions of even short duration suffice to bring back to the health and life of infants all the dangers which seemed to belong to the distant past.

The progress which has been made in the last decades in the field of infant nutrition is to a great extent based on the work of previous generations of pediatricians. It is our intention to point out the connections between the older established fundamentals of pediatrics and the many new developments in this field. Our present field of work has given us ample opportunity to gather experiences in the fields of physiology and pathology of infant nutrition. Not many colleagues in other countries are still given the opportunity to experience the pathology of infant nutrition on such a large scale. It is hoped that they will also be spared this experience in the future.

This guide is intended mainly for the practicing physician and for the student. We thus felt justified in omitting literature references. The reader specially interested in any particular subject will easily find the material in the handbooks of pediatrics.

If this comprehensive presentation will also be found of use to the specialist its purpose will be fulfilled.

L. F. MEYER E. NASSAU

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Physiology and
Pathology of
INFANT NUTRITION

PART I

Physiology of Infant Nutrition

CHAPTER I

Development of the Healthy Infant

THE BIRTH of a child marks the beginning of a period of growth and maturation which is hardly less spectacular than the development of the fetus from the fertilization of the ovum to the birth of a mature infant. The considerable increase in length and weight, the gain in knowledge and ability, the development of all organs and their functions are signs of rapid progress during this period of life. In none of the later periods of childhood or adulthood do we find a development of organs of an even comparable measure.

In every individual the final result of normal development is a combination of inherited and external factors. External stimuli of any kind in moderate strength cannot influence the path of development characteristic for the individual. With correct care and quantitatively and qualitatively adequate nutrition the development of the "Anlagen" takes place according to growth impulses. External stimuli can change very little and still less improve the results of growth and development which are conditioned by racial and familial factors. However, much can be done to impede, damage or destroy this development.* It must therefore be the task of the physician to protect the growing infant from all harmful influences so as not to disturb the natural development.

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* The results of animal experiments, which were confirmed by observations made on women who became pregnant during or shortly following internment in one of the hunger- or concentration camps, showed that malnutrition during the first months of pregnancy results in an increased incidence of malformations in the infants. Malnutrition of the pregnant woman produces the same harmful effect upon the fetus as infections (German measles, chicken pox, grippe, etc.), insufficient oxygen supply of the fetus (caused, e.g., by an anesthetic given to the mother during pregnancy), toxemia etc. On the basis of these experiences we find it possible for the first time to avoid in part the malformations of the fetus which until now appeared to be a result of a defect in the ovum or the sperm.

During the last years some biologists have emphasized that external circumstances are of decisive significance for the pattern of development, while the genes of the gametes are of less importance. These views have so far found recognition only in a few countries, and until convincing evidence in this direction can be presented the opinion that hereditary Anlage of the gametes is the decisive factor for the future development of the child will remain in force. It must be admitted, however, that living conditions, whether good or bad, can alter the developing form. Thus an accelerated development of the child has been widely reported in the course of the last twenty years. Already at the end of the first year of life we find higher figures for weight and height measurements and an earlier onset of teething than were reported some years ago. At the end of the pre-school period we find children now one to one-and-a-half years ahead of the average data reported at earlier times. The accelerated growth is attributed to improved care and nutrition and to increased amounts of light, air and vitamins of which the infant benefits nowadays.

GROWTH

The growth impulse, a hormonal force as yet not definable, stimulates the formation of new protoplasmic tissue. The growth process maintains the living tissues by continuously replacing those which die. Throughout the growth process of the child up to the end of the growth period this force also provides for new formation and changes in the tissues which increase or alter the cell masses and cell forms. At the same time some cell groups cease to function while others are put into the operation of the organism in changed or renewed form. The time of growth is limited in every individual to a certain period of life. In the human being it terminates with the completion of growth at the end of the second decade. However, further differentiation of cells, in a positive or negative sense, and cell multiplication as replacement of destroyed tissues continue throughout life.

Quantitatively and qualitatively adequate nutrition is the necessary basis for any growth process. The new formation of viable, type-specific protoplasm ceases with hunger, as soon as food intake drops below subsistence requirements. Apart from this form of growth impediment through complete external hunger, the growth of the infant can come to a standstill through partial external hunger, that is, through lack or insufficient intake of a single food component such as proteins, carbohydrates, water, vitamins or even fats. But even if food is offered in adequate amounts and quality, an internal hunger, caused by inadequate utilization of the ingested food, can result in growth impediment. But even in the most severe stages of hunger, whether internal or external, the growth impulse is not destroyed. With a removal of the damaging factors (sufficient food intake, addition of lacking food components, removal of the nutritional disturbances) the body not only compensates for the retarded growth but in many cases surpasses the expected limits in a relatively short time by accelerated growth. The process can be compared to a taut spring suddenly released. The terms potential, latent or dormant growth impulse could be applied to this phenomenon. During the period of arrested growth the formation of new cells in the tissues either stops (cessation of cell division) or cell division continues, although at a reduced rate, with the newly formed cells not reaching normal size, possibly as a result of lack of food (hypoplasia).

The human infant differs from all animals in the rate of growth and in the caloric requirements for this process. This difference from the entire animal world is characterized by the slow development and by the high caloric requirements needed for doubling the initial weight. Thus the time necessary for doubling the birth weight amounts to the following averages:

Rabbits	6 days
Dogs	8 days
Pigs	16 days
Cattle	47 days
Horses	60 days
Human beings	180 days

The total amount of calories needed for doubling the birth-weight averages in all animals is about 4000–5000. Only in the human infant this amount is increased to about 28,000 calories. Thus the consumption of milk, the most essential food during the period in which the weight is doubled, amounts in the human being to 50,000g per 1.5 kg weight gain, in all other mammals, however, to only 3000–10,000g for a similar weight gain. The growth quotient[*] in animals is around 34.4, in the human infant, however, 5.2.

What has so far been referred to as development is, however, not only a quantitative increase of a more or less complicated substance but an infinitely complex and manifold process of differentiation. There is no better way to illustrate the miracle of infant development than to compare the three stages of development through which the infant passes in the short period of one year: 1) the newborn who, in his complete helplessness, leads an almost vegetative life; 2) the infant at the age of about four to six months who has outgrown the "dumb first quarter of the year" and who, due to the development of his sensory organs, shows considerable interest in his surroundings, and 3) the one-year-old who executes purposeful movements, stands erect, walks and is capable of utilizing his senses, who speaks and understands, knows and recognizes, who can distinguish what is permitted and what is forbidden. This development and progressive differentiation can be divided into several components, some of which are clearly discernible and important for judging the child's health and will be discussed below.

GROWTH IN WEIGHT

Clear evidence of tissue growth is demonstrated in the ascending weight curve. With correct nutrition, adequate in quality and quantity, any weight gain represents increase of living substance. An evaluation of the body weight does not, however, permit any conclusions as to the type and value of newly formed tissue or the composition of the protoplasm in the newly built cells.

The first requirement for the transformation of food into protoplasm and cells is the presence of water in sufficient quantities. Therefore the food of the young, fast growing organism must be particularly rich in water. If measured by weight units the water requirements are particularly large during the time of rapid growth. The percentage of water contained in the entire organism during childhood is higher than in the period after termination of growth.

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[*] Growth quotient $= \frac{\text{energy consumption for growth}}{\text{energy consumption for metabolism}}$

Water Contents at different ages:

Fetus	97.5%
Newborn up to six months	74%
One year old	68%
Old person	60%

This water, which forms about 70% of the body weight, is found mainly within the cells (40–50%); 15% of the water is located in the interstitial tissues. The amount of water in the blood constitutes not more than 5% of the entire water reserve. These figures are of practical significance for the task of therapeutically replacing lost extracellular or intracellular fluid.

In the healthy child about one half of the water intake is eliminated by the kidneys. The water loss through insensible perspiration (skin, lungs) stands in second place, and only a small percentage of water leaves the body through the bowels (Fig. 1a). There is a close relationship between water loss through urine and through feces: if more water is eliminated through the bowels, as for instance in diarrhea, similar quantities of water are retained by the kidneys. Only if the losses through insensible perspiration are substantial do we find disturbances in this mutual relationship (Fig. 1b). If severe losses of water through lungs and skin coincide with diarrhea complete anuria may occur.

The following table indicates the intake and output of water for infants (*Butler* and *Talbot*):

	Urine	*Stool*	*Insensible Perspiration*	*Total*	*Requirements per kg Body weight*	*Total*
Infants 2–10 kg	200–500 cc	25–40 cc	75–300 cc	300–840 cc	165–100 cc	330–1000 cc

Water intake in adequate amounts has, therefore, a deciding influence on the growth process. A healthy organism eliminates excess amounts of water through the kidneys without difficulties

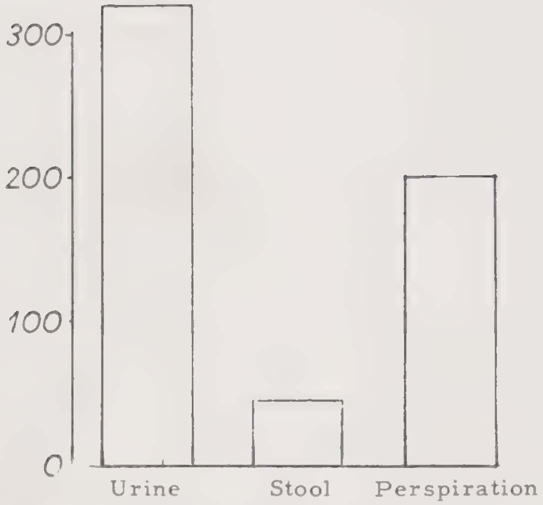

Fig. 1a. Distribution of water elimination in the healthy infant.

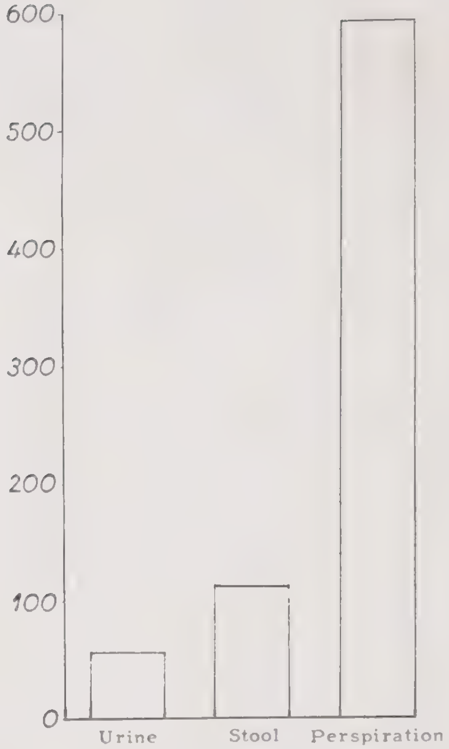

Fig. 1b. Distribution of water elimination in the infant with diarrhea.

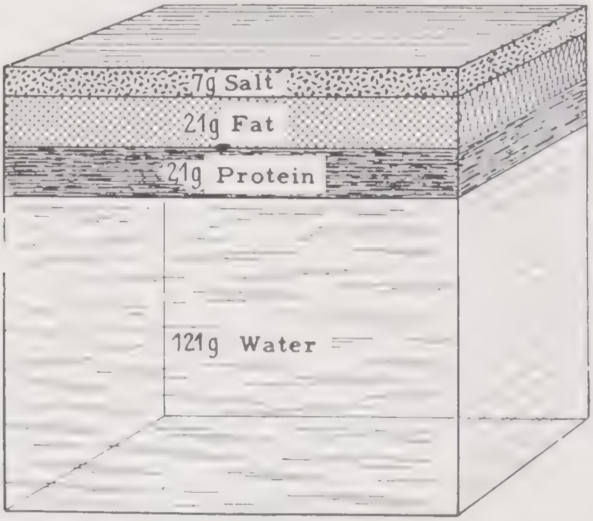

Fig. 2. Weekly growth of a healthy infant. Protein, 21g. Fat, 21g. Salts, 7g. Water, 121g. Total, 170g.

and most likely without damaging effects. Disturbances of a different nature may, however, occur in children receiving an excess amount of water (see p. 514). An excess amount of fluid intake in children with a pathologic constitution (hydropic or lymphatic constitution or children with exudative diathesis, etc.) has an additional damaging effect which can produce pathological symptoms (manifestation of the diathesis).

An analysis of daily or weekly weight increase of a growing infant demonstrates the important role of water in the growth process. With a weight increase of 25g per day the following substances are added to the organism:

Water	about 18g
Protein	" 3g
Fat	" 3g
Carbohydrate	*
Salt	1g
	25g

These figures do not indicate, however, in which places of the organism and in which form these new substances were deposited. For the weight increase of the infant is a combination of a great many factors only few of which are clinically evident in the form of fat deposits, growth of the skeleton, or increase of protoplasm (musculature). In view of the complexity of processes which take place simultaneously and which most likely influence each other, it is miraculous that the healthy infant shows as a final result a steady increase from week to week, even though the gain in body substance is not the result of the growth of the same elements in all stages of the first year of life.

Thus, despite the multiplicity of processes which contribute to the growth and despite great individual variations, it was possible to establish averages for growth in weight which apply to the majority of healthy growing infants. Moderate deviations in either direction are of no pathologic significance. The great regularity in weight gain, which causes most of the fetuses to reach a weight of 3000–3400g upon birth, continues throughout

---

* Deposited as glycogen in muscles and liver in not exactly measurable quantities, but here too 3g of water are needed for the deposit of 1g glycogen.

the first year of life. In the last fifty years an increase in the average birth weight has been observed. Only those countries hit by wars showed temporary drops in birth weights. Today the birth weight averages approximately 3300g. This rise seems to be related to the higher age of the mothers, especially in the case of mutiparae. Even under unfavorable conditions of pregnancy (disease, hunger), the children's birth weight is usually within normal limits.

The birth weight of the child is, however, greatly influenced by individual variations and racial and hereditary background. A birth weight of as low as 2500g or as high as 4500g can still be regarded as within "normal" limits. The birth weight of 99% of all newborns falls within those limits. Infants with a birth weight of less than 2500g are regarded as prematures, although this may not be justified in many cases. Children with a birth weight of over 4500g are considered giant infants. Birth weights were observed as high as 5500g and unusual cases of birth weights up to 10 to 12 kg have been reported as curiosities. Only during the second half of the first year and with increasing frequency toward the end of the first year do we observe differences which permit a classification of the infants into types such as slender, asthenic, muscular, etc.

The rise in the weight curve is temporarily interrupted soon after the transition from intra- to extrauterine life; this is called the *physiologic weight loss of the newborn.* Regular daily control of the body weight shows a weight decrease of 200–300g in the first days after birth, this weight loss being usually higher in children with a larger birth weight than in those with a relatively low birth weight. The amount of weight loss is partially dependent on the time the umbilical cord is cut. If the cord is cut later the weight loss is usually greater than if cut early. There are only few children who do not show this physiologic weight loss. In those in whom it occurs the decrease in the weight curve should not continue beyond the fourth or fifth day. Sometimes the weight curve straightens out or even rises earlier but usually only after this period of time, so that the original birth weight is again reached by about the eighth to fourteenth day of life. In the majority of children more time is needed to regain the lost weight.

A number of newborns will regain their birth weight only by the end of the third week of life (Fig. 3).

The cause for the physiologic weight decrease was incorrectly attributed to the loss of meconium, the elimination of urine, and the shedding of the remaining umbilical cord. The refractometrically proven thickening of the blood which corresponds exactly to the course of the weight curve, shows that it is essentially the water loss which causes the decrease in weight. This temporary water loss might be regarded as a withering of the newborn during the first days of life. The transition to extrauterine life forces the child which hitherto had been completely submerged in fluids and lost no water through lungs or skin to regulate by itself the complicated processes of water metabolism. Initial disturbances in this mechanism and special strain on the water household caused, e. g., by the crying of the newborn, contribute to the physiologic weight loss. A study of the respiratory metabolism proved this assumption. Metabolic tests on newborns fed physiologically with colostrum have shown no decrease in nitrogen and minerals which might explain this weight loss. Thus the cause of the decrease in weight is not tissue degeneration. Accordingly newborns fed breast milk from the first day of life lose no less weight than infants who receive only small amounts of colostrum. Only by forced feeding with breast milk or mixtures of cow's milk has it been possible to partially or completely suppress the physiologic weight loss.

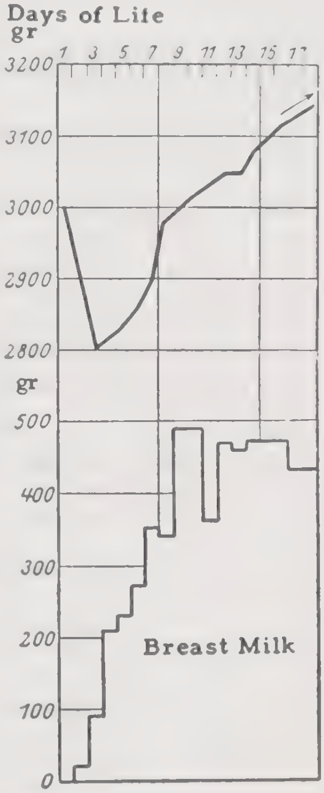

Fig. 3. Physiological weight loss in the newborn. Re-establishment of original weight after about 10 days, corresponding to slowly increasing amounts of milk.

After the initial weight has been regained the healthy infant shows a steady gain in weight. Provided the food intake is adequate, fluctuations in the progress of the weight curve are less noticeable if the infant is weighed infrequently, such as weekly or once or twice a month. If the child is weighed several times a day the fluctuations are found to be considerable and can amount to 150–300g after the first three months of life. This up and down of the weight curve in the course of 24 hours is partly due to the food intake at certain hours during the day and the long pauses during the night as customary in present day feeding schedules. Partly, however, it is due to a physiologic rhythm in the metabolic rate which, independent of the food intake, causes at times a mobilization, at times a stabilization of the water in the organism. The kidney adapts itself to these fluctuations. In the late hours of the night the organism eliminates water. These rhythmical fluctuations in the weight curve show the importance of weighing the infant always at the same hour of the day. If a child is weighed one day in the afternoon and the next day in the morning, or vice versa, the scales may show seemingly unexplainable weight losses or gains. If, for instance, a child is admitted to the hospital in the afternoon the discrepancy in weight may result in wrong conclusions and incorrect therapeutic measures. The daily ups and downs of the weight curve result in the healthy infant in the ultimate daily addition of approximately 30g of body weight. The small amount of water which is ultimately retained is shown in the following diagram by *Gamble-Pracht-Bienvenu* (Fig. 4).

If the weight is recorded at weekly intervals we find, provided the food intake is adequate, a rapidly rising curve during the first six months of life; the curve begins to flatten out between six and nine months and towards the end of the first year becomes an only slightly rising line. Only occasionally we see a renewed rise in the weight curve during the third and fourth quarter of the first year; this occurs if after prolonged and exclusive breast feeding, which at this age no longer covers the caloric requirements of the infant, other foodstuffs are added to the diet. The daily weight gain in a healthy infant averages 20 to 30g during the first half year, and 15 to 20g during the second half year; the monthly increase amounts therefore to about 600 to 900g during the first six months and 450 to 600g during the second six months of life. Upward deviations of these averages are frequent and without significance. Diminished weight gains for periods over two to three weeks demand readjustment of the diet.

> Thus we can determine the expected weight of a healthy full term infant by adding to the birth weight the age in months multiplied by 600 or 500 respectively. For instance a child with a birth weight of 3000g should at the age of three months weigh 3000 plus 3 × 600 = 4800g, and at the age of nine months 3000 plus 9 × 500 = 7500g.

The infant should double his birth weight by the age of six **months and triple it by the end of the first year.**

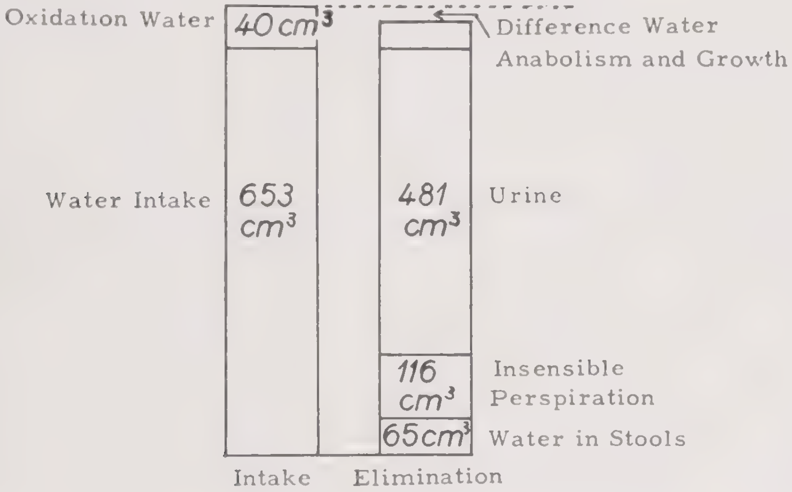

Fig. 4.

There are numerous deviations from a steady and regular weight increase: curves with a steep initial rise but early flattening, others which show a step-like increase, showing alternate progress and standstill of weight increase. Not only constitutional peculiarities are responsible for these irregularities in weight gain, but also variations and changes in the amount of food intake, constitution and temperament of the infant and climatic influences which cause, for instance, a decrease in appetite during the hot summer months. Deviations from the normal weight curve alone are not enough evidence to permit the physician to draw any conclusions as to the child's state of health. It is as unjustified to consider rapid weight increase and overweight in children as signs of strength and healthy development as it is to consider skinny children always as sickly or weak. More important than the absolute amount of weight increase is the fact that the weight gain of an infant should never come to a prolonged standstill (see chap. on Dystrophy, p. 181).

Raising the weight gain above the average values by means of overfeeding is possible only within certain limits and in certain types of children. In some children it is possible, especially during the first months of life, to bring about larger fat deposits, but new cell formation never exceeds the norm. However, we frequently find arrest in weight gain and even weight loss. They point towards disturbances of the food metabolism in the infant. For practical purposes the supervision of the weight gain is even today the simplest and also the most delicate indicator for the development of the infant, taking into account, of course, the not infrequent errors.

Weight kg	*Boys Age*	*Length cm*	*Girls Age*	*Weight kg*
3.48	Birth	49	Birth	3.24
3.7	”	50	”	3.5
3.9	”	51	”	3.7
4.1	”	52	”	3.9
4.4	1 month	53	1 month	4.1
4.7	1 ”	54	1 ”	4.3
5.0	1 ”	55	1 ”	4.5
5.3	2 months	56	2 months	4.8
5.6	2 ”	57	2 ”	5.1
5.9	2 ”	58	2 ”	5.4
6.2	3 ”	59	3 ”	5.7
6.5	3 ”	60	3 ”	6.0
6.8	4 ”	61	4 ”	6.3
7.0	4 ”	62	4 ”	6.6
7.3	5 ”	63	5 ”	6.9
7.6	5 ”	64	5 ”	7.1
7.9	6 ”	65	6 ”	7.4
8.2	6 ”	66	6 ”	7.6
8.5	7 ”	67	7 ”	7.8
8.7	7 ”	68	7 ”	8.0
8.9	8 ”	69	8 ”	8.2
9.2	9 ”	70	9 ”	8.5
9.5	10 ”	71	10 ”	8.8
9.7	10 ”	72	10 ”	9.1
9.9	11 ”	73	11 ”	9.4
10.2	1 year	74	1 year	9.7

The tables compiled by *Pirquet* with data taken from *Camerer* and *Feer* are still the best references for the average progress of weight increase of the healthy infant. During the last years a more rapid weight increase is frequently observed, especially in the early months of life, so that the child often reaches a weight of 10,000g or more by the end of the first year. It remains to be seen whether this precipitated development is of later advantage to the child or not.

The Mechanics of Growth in Weight

An attempt to describe the mechanics of growth does not lead very far. Any new tissue formation, which consists of almost 75% water, is preceded by the intake and retention of water in the body. The evenly rising weight curve of a healthy infant is essentially the result of uniform water retention. This fact is proven by the observation that rapid weight loss of several hundred grams and a similarly rapid gain within a few days is possible only if water constitutes the main factor in the increase and decrease of weight.

It is as yet unexplained how and in which form the water is absorbed by the growing and dividing cell. In any case, the method of water absorption is of decisive significance for the quality of growth and thus for the health of the child. The varying quality of absorption is demonstrated best by the reaction of the organism to damages by nutrition, infection or care of the child.

Storage of water alone is not possible in the healthy infant on a larger scale, even with extensive fluid intake. Water retention in the organism is conditional upon the mixing of water with nutritive substances.

The carbohydrates are in closest connection with the process of water retention. Animal experiments prove that the organism of some animals fed on a carbohydrate diet, who do not easily change carbohydrate into fat, such as dogs, is richer in water than the organism of animals fed on a fatty diet. The inability to gain weight on a diet lacking in carbohydrates corresponds to the lack of gain of infants fed on milk mixtures containing little or no sugar. The former failure of raising young infants on whole milk (without adding sugar) is an example for the deficient development and growth on diet lacking in sugar. Similar examples are the disturbances due to excessive milk feeding (Milchnährschaden) (p. 248), a damage caused by lack of sugar intake and corrected as soon as the organism obtains sugar in sufficient quantities. The water-storing capacity of carbohydrates is further proven by the observation that a weight gain is possible only if the food contains a certain minimum of carbohydrates. If the intake falls below this minimum, which varies somewhat with the individual, the child will not gain weight even if the intake of all other food components is considerably increased. The water retention of carbohydrates results in an improved retention of N, Cl, and Na and K. The carbohydrate requirements are further influenced, apart from individual variations, by the composition of other food components. Mixtures rich in whey require less addition of carbohydrate than foods containing only little whey. Temporarily some of the salts (Na, Cl and K) can compensate for a lack of carbohydrate. Food mixtures rich in protein and fat often require less carbohydrate than others, since the organism of the infant can build carbohydrates from a surplus of protein and fat, although only to a limited extent. The age of the child is also of significance for the amount of carbohydrate requirements. Young infants, in days of health or in compensation for pathologic disturbances, need relatively more carbohydrates than children in the second half of the first year.

Any addition of carbohydrates results in a considerable weight gain. For this gain the addition of even a few grams of sugar may suffice. The weight gain is lost again if after some days the additional carbohydrates are discontinued. A control of the urine secretion under various conditions proves that it is a question of absorption and elimination of water through carbohydrates. By adding flour and sugar to the food the urine secretion will be reduced by almost half in many children. In the so-called combined water experiment (intake of sugar water) the hydropigenous quality of carbohydrates is shown by the small and delayed quantity of urine secretion (Fig. 5).

The various carbohydrates act differently with regard to the ability of absorbing water. The reasons for these varying effects

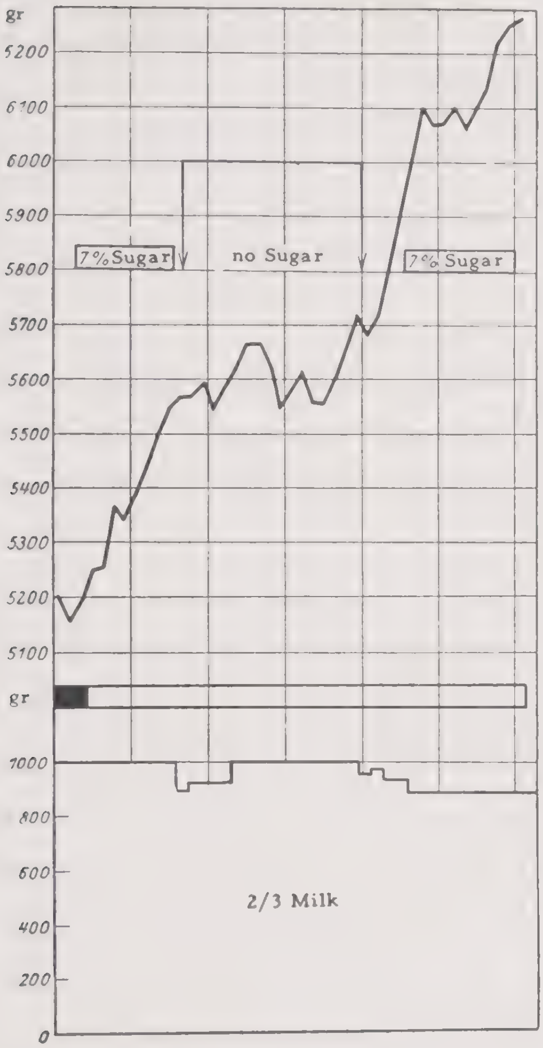

Fig. 5. Weight increasing action of carbohydrates. Six months old infant. The omission of sugar from a $^2/_3$ milk diet during a time of good weight increase results in immediate arrest of weight gain despite isocaloric increase of fat in the diet. Renewed addition of sugar again produces weight increase. Stools formed during the entire period, at a rate of two to three times a day.

lie partly in the differences in digestion of flours and sugars in the intestines. Certain by-products which appear in the process of roasting, baking, carmelizing or dextrinizing of various types of flour or sugar may also be of significance. Finally the colloidal composition of the feeding mixtures influences the amount of water absorption. The addition of mucilage to amounts of sugar which just cover the requirements has a more favorable effect on weight gain than the addition of calorically equal quantities of crystalline sugar.

Very little is known about physico-chemical processes in water retention through carbohydrates. The storing of part of the carbohydrate as glycogen, which requires three times its weight in water for its deposition, explains only in part the considerable weight gain which is clinically observed after adding carbohydrates to the diet.

The effects of the water binding forces of salts are somewhat better known. The water absorption through salts and carbohydrates is not of the same value in its mechanism and in its significance for the organism. The water retention which takes place through carbohydrates is more stable and equals more real tissue formation than water retention through salt intake. The result here may be a pre-edema or even a real edema. As proof for the different actions of carbohydrates and salts with regard to water retention we can mention the observation that upon the discontinuance of previously added carbohydrates the weight curve usually does not return to its initial level. Part of the water absorbed in the tissues is already retained in a form which remains absorbed despite lack of sugar or flour. By the addition and subsequent removal of hydropigenous salts (e.g., NaCl) the weight curve returns to its initial level.

Na, K and Cl are of decisive significance for water retention in the organism. The distribution of the various ions of the salts in the organism is not even.

Water and salts are stored in the organism in three large deposits: 1) in the fluids contained in the blood vessels (plasma-erythrocytes); 2) in the intercellular fluid, and 3) in the intracellular fluid. Each of these reservoirs is separated from the other by semi-permeable membranes through which, to a certain extent also in the healthy infant, the ions of the salts and thus also the Na and K can penetrate in either direction. Changes in the salt content in one part of the system lead to the migration of ions into other parts in order to keep osmotic pressure and reaction of the fluids as constant as possible. The majority of Na ions are found in the plasma and in the intercellular fluid; sodium is the salt of the extracellular fluid. The major part of K, however, is contained in the intracellular fluid. Changes in the electrolytic concentration in the cells through cellular loss of potassium in quantities exceeding the normal exchange of ions lead to functional disturbances of the cells (see below).

Only the free ions of the salts can influence the water metabolism. Cations (Na, K) are more water binding than anions (Cl, HPO_4, HCO_3, etc.). The calcium ion acts strongest in withdrawing water or in preventing water absorption. The water binding power of sodium chloride was formerly attributed to the chlorine ion. But also in the adult we consider today the sodium of prime importance for water retention, although only if other reasons for edema exist. By comparative experiments with different sodium salts it has been proven that the anion, too, influences the amount of water retention. It has been shown that the strongest water binding forces are in the chlornatrium.

The quantities of NaCl required for normal absorption are relatively small (see Fig. 6). In calculating the sodium chloride metabolism of healthy individuals we find that 90% is eliminated in the urine and only 6% in the stool. About 3% of the ingested sodium chloride is retained for anabolism.

Water retention due to addition of salts can be observed only under certain conditions. Minor salt increases will hardly result in major changes of the weight curve in a healthy infant. Water retention can be seen clearly after daily addition of 2 to 3g of sodium chloride in infants during the first weeks of life or in older infants with chronic nutritional disturbances. In either case salt elimination is less than intake. Retained salt must bind water in order to maintain the internal osmotic equilibrium. Water retention does not become evident in the older child or in the adult even after intake of larger amounts of salt, because the salt is quickly eliminated as long as the kidney function is normal.

Only by weighings within short intervals after salt intake do we observe the water retention by the salt in form of temporary weight increase.

For normal functioning, particularly of the vegetative nervous system, a certain correlation of the salt ions is of significance.

Carbohydrates and salts participate only indirectly in the water absorption of the organism. They are the driving forces which effect the water transport to and from the tissues. However, the protein too, seems to have a direct influence upon water retention. At least observation and the result of metabolic tests speak for it. Addition of protein preparations to the food of poorly thriving breast fed infants and also some bottle fed infants results in an immediate weight increase which can at least in the beginning be explained only by water retention.

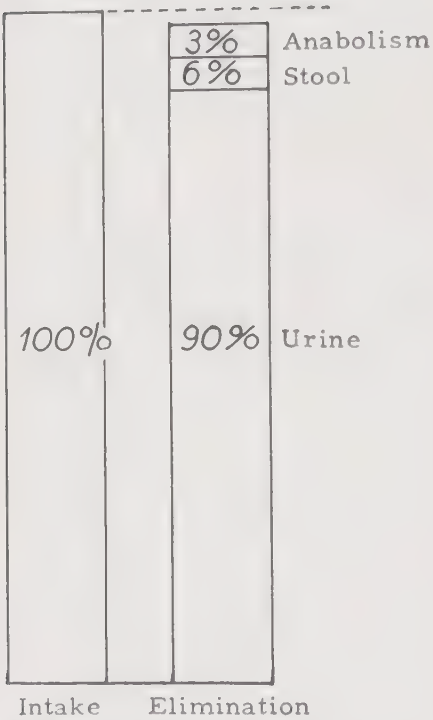

Fig. 6. Intake and elimination of sodium chloride in the healthy infant.

The real water binding substances through which water is retained in form of protoplasm are the colloids. The osmotic pressure of the colloids which are present in the cells in the form of so-called micellae[*] directs the flow of liquid in the organism. Differences in osmotic pressure between serum, intercellular fluid and cells (plasma of cells) cause movements of fluid. These differences can be due to local changes in the osmotic pressure caused by variations in reaction, migration of ions of salts (see preceding page), hormones or drugs.

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[*] The living substance is believed to consist of so-called micellae. Micellae are protoplasmatic particles composed of more or less numerous molecules which are held in suspension so that they no longer obey the law of gravity. The sum total of these particles determines the colloidal state of the living substance. Micellae are surrounded by a thin, adsorbing, perimicellar layer which divides the particles and holds them in suspension. In the young individual the micellae are smaller, their total surface, therefore, larger and they have the capacity to strongly adsorb water. This water adsorption increases the osmotic pressure in the cells and thus stimulates cell division.

The osmotic pressure of the colloids which remains constant in the adult human and the adult animal, approaches this value only gradually during the period of growth. We therefore find that young tissues have a greater ability to swell than the same tissues in older individuals. A cessation of swelling or deflation of cells results in stoppage of growth or weight loss, a phenomenon which is of interest for the pathology of infant nutrition.

Further substances in the food which aid in the water retention are the vitamins. The action of vitamins upon the growth process depends upon changes of the colloidal structure of the protoplasm. Vitamin intake is believed to cause peptization, that is, stronger dispersion of colloidal particles, which as a result of the enlargement of their surface can adsorb more water. Futhermore, some authors have attributed to the vitamins A and B a role in the formation of growth promoting hormones in the thymus. Perhaps here, too, a change in tissue reaction is of significance. Vitamin deficiency causes acidosis, vitamin intake alkalosis. Certain vitamins cause acceleration of cell respiration and cell oxidation. But the influence of vitamins upon the mineral metabolism, which has been proven also in the infant, is of great significance for the water metabolism and thus the growth process of the child.

Fats are not believed to have any positive connection with water absorption, since the fattiest tissues in the organism contain the least water. Apparently all types of fats show this negative relationship to water storage. However, those fats which contain lipoids and phosphatides seem to contribute to the firmness of the tissue structure. As a result of their surface activity they lend strength to the protein structure.

A survey over the significance of the various nutrients for the growth process shows the water adsorption on colloids as the core of the problem. These osmotic processes are the basis for cell growth and cell division. The processes of growth and water retention are inseparable. The main premise for normal water retention is the presence or the formation of a sufficient quantity of type specific protein whose osmotic quality is regulated by influences originating from salts, carbohydrates and vitamins. Apart from this a number of hormonal influences (hypophyses, adrenal cortex, etc.) and the tissue reaction are of significance. Carbohydrates and salts proper do not participate actively in the healthy individual in the processes of water retention. They are forces promoting cell swelling which are capable of attracting water to the growing cell and the colloids.

The antagonism between sodium and potassium salts, carbohydrates and vitamins on the one side as forces promoting cell swelling and calcium salts and fats on the other as forces restraining cell swelling is, apart from all theoretical considerations, also of practical significance in the choice and composition of infant nutrition. Food which causes excessive water retention is as undesirable for the infant as food which through its composition prevents water absorption and growth: for normal weight gain, therefore, we need a combination of all food components in certain correlation. Only a complete chain of food substances can assure optimal tissue formation even in the artificially fed child.

These conceptions, however, also show the peculiarities of the process of osmosis in the growing infant. Whereas the growth process in non-living colloids and partly also in plants consists only of storage of water and salts, the procedure in the infant involves complicated processes in the protoplasm. Compared with the plain gel or even the plant, the loss of water of the tissues in the infant, that is, a weight loss, is, therefore, not a simple reversible process. Every weight loss is always connected with considerable, sometimes nonreversible changes and damages of the living substance.

The selection or composition of food for the infant might appear as an unsolvable problem in view of these diverse effects of the different food components upon growth. Breast milk represents in quality and quantity of its composition an ideal food which covers the needs of the growing infant in every direction.

This finding has repeatedly led to the attempt to imitate the chemical composition of breast milk in quality and quantity of its components. These efforts have so far not been successful, despite all progress in science and technology. Since we have in the meantime, however, succeeded with steadily increasing success in raising infants on various artificial food mixtures we must ask ourselves where, in the last analysis, the secret for the wonderful effect of natural feeding lies and which prerequisites must be fulfilled in order to approximate heterospecific milk or milk mixtures to the natural food of the infant (see chapter on Milk).

Evidence of solid water absorption in the healthy infant is the good tonus of the musculature, the good turgor of the tissues, the beautiful shimmering of the eyes and the good circulation in the skin. The capacity for regular water absorption is, however, not present in all infants to the same extent. With similar nutrition and despite prevention of more serious faults in food composition and intake, the physician will always find children whose capacity for good water absorption is not adequately developed. The result is an abnormal and pathological tissue formation. The ability for normal solid water fixation is called hydrostability; the opposite, a pathological, loose water fixation is known as hydrolability. Other authors speak of a hydropic constitution. The two terms do not seem to express quite the same: while a hydropic constitution signifies the peculiar tendency to accumulate loosely bound water which in infections etc. is just as easily given up, the term hydrolability does not necessarily include the concept of above-normal water absorption. Hydrolability signifies first of all the absence of physiologic solidity of water fixation. Water absorption itself, however, does not need to exceed the norm—and this probably also corresponds to the clinical fats. Hydrolability shows itself in a constant up and down, in considerable fluctuations of the weight curve which appear in these children for minor causes and which prevent a steady weight gain. The history of the infant often aids the physician in judging its capacity for water fixation (see also p. 514).

The basis for the physico-chemical peculiarities of abnormal water fixation is not known. This pathological condition is influenced by nutrition. The tendency to retain water on a one-sided nutrition, rich on carbohydrates (damage due to excessive flour consumption—"Mehlnährschaden") might here be mentioned. The significance of the constitution is shown by the fact that some children show signs of hydrolability, without nutritional disturbances, even if fed on breast milk, while other children, fed artificially, remain hydrostabile, despite faulty nutrition. Peculiarities of the endocrine glands (hypophysis, adrenal cortex) may be of significance. In the final analysis the cause would probably be found in disturbances of the central nervous system.

GROWTH IN LENGTH

Among the multitude of growth processes the process of growth in length is of special interest. A control of this accomplishment in the child is just as important for determining the state of health, even more important, according to some authors, than regular determination of body weight. Next to the scale the tape measure belongs to the pediatrician's tools.

Growth in length is above all the expression of skeletal development. Observation of growth in length makes it possible to pursue in a single system of the organism the influence of internal and external factors upon normal and pathological processes. The development of the bones consists of growth in length, growth in weight and differentiation. These processes run parallel according to a prescribed plan. Dissociation of the processes is, however, possible. In case of myxedema, for instance, differentiation lags behind weight growth. A sign of progressing differentiation is the appearance of bone centers in the cartilagenous epiphyses of long bones. The appearance of bone centers in the course of the first year of life does not follow a strict course, as previously believed. Variations are possible in the sequence of appearance of the bone centers. Present at birth are: the ossification center of the proximal tibial epiphysis (the absence of which at time of birth is considered to be a definite sign of prematurity), the bone centers of the talus and the cuboideum and the bone center of the lower femural epiphysis. In the fourth month of life the bone center of the capitate appears, between the first and sixth month that of the hamate, between the fourth and eighth month of life the centers of the proximal humeral epiphysis, in the sixth that of the third cuneiform and in the tenth that of the head of the femur. Simultaneously the tubular bones undergo a change in their inner structure. The more fibrillar structured bones of the newborn, which are rich in large bone particles, blood vessels and bone marrow elements, develop in the first year of life into the firmer structures of the adult bones, arranged in accordance with the requirements for weight bearing. The mechanical utilization of the bones through muscle pull is essential for this change in bone structure.

The intensity of growth in length varies with age. It is strongest during the period of fetal life during which the fetus grows in nine months from the egg to the newborn with a length of 50 cm. Growth in length still maintains its intensity during the first three months of life during which the length of the infant increases by over 8 cm. Towards the end of the first year of life growth in length slows down but does not stop, except for very short spans, during the entire period of childhood and adolescence.

The newborn infant measures at birth 49 to 52 cm in length. Boys usually are longer at birth than girls. Through reshaping of the deformation of the head skeleton, caused by the passage through the birth canal, and the disappearance of edema of the scalp, we occasionally find after birth a decrease in length which can amount up to 1 cm. The true length of the newborn can therefore be determined only on about the tenth day of life. From this point on, the length of the healthy infant increases steadily although not regularly. The rapid growth spurt during the first weeks and months of life, reaching its maximum during the second month, is followed by a slowing down of growth during the second half of the first year. The average monthly increase amounts to about 2 to 2.5 cm in the first half year and to about 1 to 1.5 cm in the second half year of life. Static pressure on the spinal column at the onset of sitting and standing simulate a slowing down of growth towards the end of the third quarter of the first year.

The amount of length increase varies with the different months of the year, although this is less apparent during infancy than during early childhood. In the northern hemisphere it is greatest in the months April to June, decreases during the hot months, shows a slight increase between September and November and again decreases during the winter months. For every period of life there are certain correlations between length and weight:

at birth the proportion is 60g in weight for every centimeter in length, at the end of the first year it is 125g. These figures show that the progress in length proceeds along a pattern which is independent of weight growth in a narrow sense.

A study of length increase in a large number of healthy children as calculated from the data of several publications, shows the following averages for the first year of life:

Length Increase Per Month	*Length Increase Per Quarter*	*Expected Length*
		at birth 50 cm
1st month 2.8 cm	First quarter	End of 1st month 53 cm
2nd " 3.5 "	8.4 cm	" " 2nd " 56 "
3rd " 2.1 "		" " 3rd " 58.5 "
4th " 2.2 "	Second quarter	" " 4th " 61 "
5th " 2.1 "	5.9 cm	" " 5th " 63 "
6th " 1.6 "		" " 6th " 64.5 "
7th " 1.5 "	Third quarter	" " 7th " 66 "
8th " 1.1 "	4.0 cm	" " 8th " 67 "
9th " 1.4 "		" " 9th " 68.5 "
10th " 1.1 "	Fourth quarter	" " 10th " 69.5 "
11th " 1.2 "	3.4 cm	" " 11th " 71 "
12th " 1.1 "		" " 12th " 72 "

For prematurely born infants the length measurement is a useful aid in determining the degree of prematurity:

45 cm length corresponds to	9 weeks of prematurity
46 cm " " "	3–4 " " "
47 cm " " "	2–3 " " "
48 cm " " "	1–2 " " "
49 cm " " "	1 " " "
50 cm " " "	full term

There are, however, numerous deviations from the norms which can appear even without external disturbances and which must be caused by constitutional variations. In any case, a length increase at a more rapid pace than that of the norm does not mean a better development in other fields as for instance in the development of static functions, just as, on the other hand, a retardation of length growth need not always point toward a slowing down of other functions.

Although regular control of gain in length is considered by some authors as more important in judging the degree of development than the determination of the deceptive body weight (fat, edema, etc.) we must also be aware that one-sided observation of growth in length can give rise to misinterpretation.

Cessations of growth in weight and length do not always run parallel. Thus an atrophic infant of three months can show a normal body length of 59 cm and a weight of perhaps 3.5 kg (instead of 4.8 kg). Such dissociations between growth in length and in weight are found primarily in young infants during the first months of life. The great intensity of the growth impulse at this young age is not arrested even by serious nutritional disturbances. Cessations of length growth for longer periods are in the infant always a sign of serious damages of the tissue-building functions. Even in severe cases of acute diarrhea, accompanied by prolonged interruption in weight gain, we find undisturbed increase in length. Only considerable and longer lasting malnutrition (as in stenosis of the pylorus) can damage the growth impulse at this young age. This situation, however, changes with the second half of the first year. Any infection, any longer lasting interruption in weight gain can then suffice to arrest the growth in length. But here too, we occasionally find exceptions to the rule. Undernourished children, being forced to live in dark shelters in times of siege, showed even precipitated increase in length, similar to plants which start sprouting in basements without light, and grow more rapidly than the same plants grown under normal conditions. The delay or interruption of growth in length at any age, however, lasts only as long as the damage which caused the stoppage. Upon removal of the damage, growth is resumed at increased pace. The growth impulse endeavors to make up for lost time and to reach its goal. Absence of sufficient growth following apparent clinical recovery demands caution in the prognosis of the disease. Even under favorable conditions it is not always possible, after considerable lag in length growth, to make up for the loss by the end of the first year.

A stronger retardation in weight increase as compared to length increase, caused by light or moderately severe disturbances, is of practical significance in the determination of food requirements. The usual tables for determining nutritional requirements are based on weight units. A certain quantity of food is calculated and ordered for every kilogram of body weight (calories). If a child suffered considerable loss of weight due to illness, so that we find in place of a well rounded body a thin and relatively too long one, it is doubtful whether the amounts of food which correspond to the reduced body weight really cover the requirements of the child. If the weight loss of the infant has not been excessive it would be more appropriate to calculate the nutritional requirements on the basis of length and its corresponding expected weight. "A child's age is measured by its length and not by its weight." The determination of nutritional requirements in malnourished children, therefore, cannot be done on the basis of body weight alone (see Nutritional Requirements).

In addition to the body length, the regular determination of growth of head and chest circumference is of practical significance in the judgment of the development of the infant. In order to avoid errors in the determination of chest circumference it is best to have the child always in the same position, that is, lying down, when taking these measurements. In order to obtain comparable values, the chest circumference is measured at the height of the mammillae, in a position of the thorax between inspiration and expiration. The head circumference is measured at the point of largest circumference, that is, usually at the protuberantia occipitalis and the frontal bosses. The measurement of the head circumference soon after birth, which might have caused dislocations of the skull bones, caput succedaneum or cephal hematoma gives inexact figures. The determination of the abdominal circumference is practically without significance. It is subject to so many errors (adipose tissues, fullness of stomach, air contents of stomach and intestines) that it is impossible to draw conclusions from these figures regarding the development of the infant.

The increase in head and chest circumference is not only a result of growth of the bones. The changes of the cranium and the thorax are, apart from basic growth processes, conditional upon anatomic and functional changes of the organs which they enclose. The head, measured at its largest circumference, grows considerably in the course of the first quarter, with a subsequent slowing down:

Head circumference in the first month of life 35 cm (32–36)
” ” ” ” third ” ” ” 41 ”
” ” ” ” sixth ” ” ” 43 ”
” ” ” ” ninth ” ” ” 45 ”
” ” ” ” twelfth ” ” ” 46 ”

Slight deviations from these figures are often found even in healthy infants. Marked delay in head growth and premature closure of the fontanels take place in cases of retarded brain development (microcephalus) or in cases of primary premature synostosis of the skull sutures. The skull bones become exceptionally hardened, the sutures appear narrow in the roentgen picture, prematurely ossified or already closed. A very small head circumference can occasionally be found in a normal child. In such cases the head form is more important than the head circumference since in pathological microcephalus the occiput is missing and the skull, therefore, flattened out. On the other hand hydrocephalus or the large brain, rich in water and poor in lipoid and calcium, which is found in children with rickets, leads to more rapid skull growth. In prematures, too, the head grows much faster in relation to the rest of the body.

The thorax not only shows an increase in size during the first year of life, but also a change in form. The barrel-shaped thorax of the young infant, with the ribs standing off almost horizontally (angle of ribs and spinal column 70 to 90°) has a larger sagittal diameter than transverse diameter. Only in the second half of the first year does the transverse diameter overtake the sterno-vertebral diameter due to rapid growth of the lungs and the transition from abdominal breathing, peculiar to the newborn, to thoracic breathing. Simultaneously we find a lowering of the ribs and thus the form of the thorax of the infant approaches that of the adult at the end of the first year.

Discrepancies of the growth of the skull in comparison with body development are practically not known in the infant with the exception of rickets and a number of cerebral processes (hydrocephalus). The size of chest circumference, however, follows more or less the course of the weight curve. Interruption of body growth is accompanied by interruption in the growth of thorax circumference. Even a decrease in chest circumference can be observed in serious nutritional disturbances as a consequence of reduction of fat, musculature and through loss of tissue fluid. In the course of recovery, the chest circumference, too, begins to expand again.

The relationship of chest circumference to head circumference is of practical significance. The head circumference of the healthy infant, especially during the first months of life, is larger than the chest circumference, provided both measurements are taken in a lying down position. There are occasional exceptions, especially in large newborns with a high birth weight, where head and chest circumference can be of the same size. Only toward the end of the first year of life do head and chest circumference approximate each other, or the size of chest circumference might even exceed that of the head. Excessive growth of the head in the second half of the first year is always pathological. It indicates at an early age the beginning of hydrocephalus, around the sixth month of life frequently the beginning of rickets.

CHEST MEASUREMENTS OF THE INFANT

		Boys			*Girls*	
	Circumference	*Sterno-vertebral Diameter*	*Transverse Diameter*	*Circumference*	*Sterno-vertebral Diameter*	*Transverse Diameter*
Newborns	32 cm	7.7 cm	7.2 cm	31.2 cm	7.5 cm	6.9 cm
3 months	37.5 "	9.0 "	8.8 "	36.9 "	8.6 "	8.7 "
6 "	40.3 "	9.3 "	9.7 "	38.9 "	9.0 "	9.0 "
9 "	43 "	10.0 "	10.4 "	41.8 "	9.6 "	9.9 "
12 "	45.8 "	11.3 "	11.3 "	44.5 "	10.5 "	10.5 "

DEVELOPMENT OF ADIPOSE TISSUES

The development of adipose tissues in certain amounts is, apart from increase and differentiation of protoplasmic substance, an important function of the growing infant. The adipose tissues, which give the healthy infant its beautiful rounded body form, are absent at the time of birth. Already at time of birth we can differentiate between skinny children with only 2 to 2.5 mm of abdominal fat or less, and fat infants with 5 mm or more. These differences, however, do not permit conclusions as to the constitution or later development of the child. It is also probable that part of the tissue which gives the newborn his fat and round appearance is not fat but water storaged in the subcutaneous tissues.

> The measuring of adipose tissue is done with a vernier gauge, similar to the one used by the precision mechanic, with which a raised skinfold is grasped. The reading in millimeters does not correspond to the absolute thickness of fat. It is composed of the double thickness of adipose tissue plus double thickness of the epidermis (about 2 mm) plus double thickness of the subcutis. In addition there are inaccuracies caused by the changing water contents of the subcutis. For practical purposes, however, and despite all inaccuracies, the figures read on the vernier gauge suffice for comparison of development of adipose tissue.

Even apparently fat children lose part of their weight during the first days of life. Prematures are particularly skinny. The fat deposits, which are present in most full term infants, are stored only in the last months of pregnancy. The real panniculus adiposus is acquired only in the course of the first six months of life. The presence of adipose tissues in the infant is a sign of health.

Which food components contribute to the fat formation of the young child? According to physiologic principles, fat can be formed from an excess of food, from fats, carbohydrates and also protein. From the point of view of nutrition a fat formation from protein is practically out of question. The possibility of fat formation from carbohydrates is proven for the organism of animals by the experiences of farmers and animal breeders. But even among animals not all of them show the same reactions. We find on one hand animals which can easily be fattened on carbohydrates (pigs, geese), and on the other hand those which cannot be fattened at all or only with great difficulty (cats, rabbits). In the adult human being fat formation from sugar has been proven by the considerable increase of the respiratory quotient caused by food rich in carbohydrates. The infant probably shows a similar reaction. The practice of infant nutrition shows that not all infants are capable of forming fat from carbohydrates on a fat poor diet. The majority of infants will remain thin on a diet containing little fat and much sugar (e.g., buttermilk—flour—sugar), even if the weight gain is satisfactory. Only after fat is added to the diet will the children begin to form adipose tissues. It is therefore incorrect in the majority of infants to consider fat and carbohydrates of the same value with regard to fat formation.

Fat formation from fats contained in the diet has been proven. In measuring the respiratory quotient on a fatty diet we do not obtain the values which might be expected if the fat in the organism would be completely burned. Hetero-specific fat with its qualitative peculiarities could be traced in the fat deposits. Goose fat, linseed oil, palmin, oil of sesame, olive oil, which had been contained in the food of mother or infant, appeared again unchanged. The qualitative permutations which the fat deposits undergo in the course of the first year (see below) can perhaps be explained by such peculiarities of the fats contained in the diet. The fat which is absorbed from the food into the organism turns always into depot-fat only. The formation of vital fats and fatty substances (cholesterin, lecithin, phosphatide), essential for the activity of cell membranes, for processes of immunity etc., is, on the other hand, brought about as a result of a complicated process of the intermediary metabolism.

For practical purposes it is obvious that any artificial infant nutrition must contain fat; the composition of the natural food points in the same direction. The composition of all accepted feeding mixtures corresponds to this requirement. The advantage of fat deposits lies only partly in the accumulation of a food reserve. Apart from that the fat layer in the skin contributes to economies in heat dissipation, since the surface of an infant body rounded by adipose tissue is smaller relative to its weight than that of an infant of the same size but skinny. Above all the state of immunity seems to be better in a body nourished on a fatty diet than in a child raised on a diet containing little fat. Fat gives the organism special nutritive substances, among them vitamins A and D which are lacking in other foodstuffs and which the cell is unable to build up by itself.

In the growing child the deposition of fat follows certain rules. In the newborn the deposit of adipose tissue is best seen in the face. On legs, arms, chest and shoulders adipose tissues are scarce. On the abdomen fat is lacking almost completely in the majority of newborns. If there is a deposition of fat in the first weeks of life it is deposited first in the face. The development of adipose tissues on arms, legs, chest and back follows. Only after all these fat deposits have formed we find after the sixth week of life a gradual formation of abdominal fat. The development of abdominal fat is more regular and stable than the development of the other fat deposits, provided there are no growth disturbances. Chemical characteristics of the fats in the various parts of the body are responsible for this. The progress of development of adipose tissues reaches, in the majority of artificially fed infants, its peak around the sixth month of life. Between the sixth and seventh month we find often an arrest in the fat deposition. In its place we observe a better development of musculature, corresponding to the beginning of development of static functions around that time.

The amount of adipose tissue in the most important periods of life can be demonstrated by the following figures:

HEALTHY CHILD

	Adipose Tissue			
	Face	*Thigh*	*Abdomen*	*Upper arm*
Newborn	7 mm	7.7 mm	3 mm	4 mm
3 months	14 "	13.1 "	5 "	6.6 "
6 months	17 "	19.2 "	8 "	9.3 "
1 year	18 "	19.6 "	8.8 "	11.3 "

Only children whose development is undisturbed will accumulate fat deposits of normal size in the different parts of the body. The presence of all fat deposits is, therefore, an important criterion in the health of the child.

The fat deposits in the organism of the infant also differ in quality in the various parts of the body. Besides, there are constitutional variations in the composition of fat. The firm and granulous fat, which is characteristic for all newborns, develops in the first months of life into two types of fat, different to touch: the raised fat fold feels solid and firm, or it is of the same thickness but withered, slack, apparently filled with fluid, and pasty. The solid fat is normal for the infant. Pasty fat points toward the presence of a constitutional anomaly. Pasty fat disappears very easily in case of infection. So far it has been impossible to find chemical differences between solid and pasty fat. Perhaps these differences do not lie in the composition of the fat but in the character of the connective tissues.

> The recognition of the differences in the two types of fat and their clinical evaluation are not new, as shown from the following quotation from the year 1751: "If the adiposity is ex Dispositione hereditaria, and the children show firm and solid flesh, then they can be like others regarded as healthy; if, however, the adiposity comes from the milk, and the habitus corporis is withering and slack, then it should not be regarded as healthy fat and flesh; such children are subject to many diseases and peculiar diatheses; their skin gets easily raw, they develop narrow chests, coughs, diarrheas and eczema, and when they are weaned this milk-flesh breaks down and they can easily become subject to atrophy or rickets" (*Johann Storch*).

The chemical composition of the fats in the fat deposits changes in the course of the first year in accordance to a pattern. The fat of the newborn differs from the fat of the adult by its relatively high palmitic acid content:

	Child	*Adult*
Oleic acid	67.75%	89.80%
Palmitic acid	28.07%	8.16%
Stearic acid	3.28%	2.04%
Free fatty acid	3.21–5.15%	1.50%

In addition, the fat of the newborn contains more water and less olein than the fat of older infants. In the course of the first year of life that fat undergoes a "maturation process," whereby the palmitic acid gradually decreases and the oleic acid gradually increases. The fat of the older infant no longer appears granulous and firm but wax-like or almost semi-liquid. Added to these differences are variations in the different fat deposits. In the newborn the fat of the various parts of the body is still of the same chemical composition, so far as can be determined from the iodine number. The fat of cheeks and thighs show later on a higher iodine number than the abdominal fat. Abdominal fat contains less oleic acid and is therefore firmer than the fat of cheeks and extremities. The firmest fat is found on Bichat's fat pad of the cheek. The composition of adult fat is reached only at the end of the first year of life. Differences in the solidity (meltability) cause differences in the reduction of fat deposits under the influence of infections or nutritional disturbances or hunger (see below).

DEVELOPMENT OF ORGANS AND PROPORTIONS

Growth in weight and in length are only the most obvious signs of normal or pathologic development. Simultaneously and parallel, partly independent of each other, run a number of other processes which combine to raise the infant from the stage of a newborn to the advanced level of the one year old. In the description of the development of adipose tissues, a factor, definable by simple means, has been selected from the multitude of biological happenings and described in more detail. This example has been chosen because—as will be shown later—the presence or absence of adipose tissues is of special significance in judging the nutritional state of the infant. Far beyond that, however, we find in the course of the first year in every cell system, and perhaps even in every cell, far reaching changes, alterations and new formations which today can be determined only to a small extent anatomically, functionally or clinically. The sum of these processes corresponds to the maturation of the infant body. The period of the first year of life brings such a rapid development and expansion of the organs present at birth that it almost equals the fetal development in diversity and speed, as has already been stressed in the introduction. But here, too, we can observe a slowing down of post-natal development, progressing from month to month. The essential, growth-delaying element lies in differentiation.

Growth and differentiation of the organism are evident by a change of its proportions. The body proportions of the newborn differ characteristically from those of the small child and even more from those of the adult. The measure of the farthest distance of the horizontally outstretched arms and fingers is shorter than the length of the body; sitting height, that is, the measure-

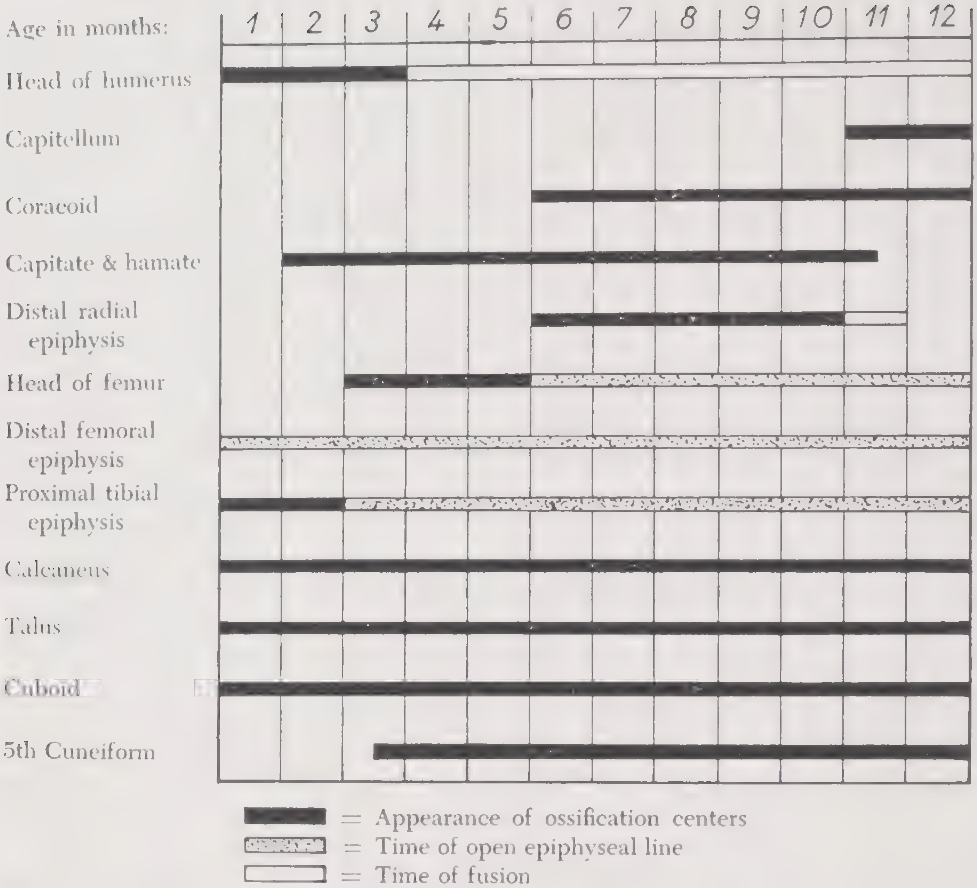

Fig. 7. Appearance of some bone centers. Time of the open epiphyseal line and time of fusion. (Great individual variations of these processes must be pointed out.)

ment from crown to sciatic bone and also the length of the trunk are greater than the length of the legs. The trunk is longer than the arms and head circumference is larger than chest circumference. From these data emerges the form of the short-legged, short-armed, compact infant body, with a short neck and relatively very large head. Especially the head, which is very large in relation to the rest of the body, emphasizes the appearance of roundness. Whereas in the adult the relationship of head to total length is 1:8, it is 1:4 in the newborn and in the child at the beginning of the second year of life it is already 1:5. The face, however, is relatively small. The proportions which differ from those of the adult are caused by the exceedingly large skull. The form of the chest also increases the rounded compactness of the infant body. The transverse and sagittal diameters of the chest are almost equal. Only with the appearance of the first static functions does the chest undergo essential changes in form caused by the changing breathing technique. The transverse diameter of the thorax increases so rapidly in relation to the sagittal diameter that the chest assumes a flatter but broader appearance. This flattening out is caused by a lowering of the ribs which are no longer almost horizontal (during inspiration) but more oblique, running from a higher position in the back to a lower point in front. The lungs adjust to the changed form of the thorax and move downward, while simultaneously trachea and bronchi increase considerably in length. The flat, almost kyphotic vertebral column of the young infant shows toward the end of the first year of life and still more during the second, the first signs of the curvatures as we find them in the older child. Toward the end of the first year of life we see that the muscle groups and bones begin to become more visible on the rather shapeless back of the young infant. The differentiation of bones is apparent in the closing of the fontanel and the changes in relationship of the length of the extremities to the length of the trunk. It coincides with a maturation of the bones in their finer structure, the practical and important expression of which is the appearance of new calcified bone centers in the cartilaginous bones. The beginning calcification of certain bone centers at certain times can be used as an approximate measure for determining the age during the first year of life (see p. 26 and Fig. 7).

Regarding the development of the various tissues we shall mention only what might be of practical significance, in the first place the changes in the blood. The initial high leukocyte count in the blood decreases gradually. The neutrophylia during the first two days of life is followed by a period of relative lymphocytosis of about 70%, which by the end of the first year diminishes to about 50–60% lymphocytes. The abundance of lymphocytes in the blood of the child corresponds to the abundance of lymphatic tissue. The number of lymph follicles in the lymph nodes and in the spleen is large, while the connective tissue is almost absent in the beginning. In striking contrast is the lack of development of lymphatic organs in the throat, mainly of the tonsils of the young infant, which are not visible at all or emerge only very little from the niche of palatal arches. The absence of tonsillitis in this period of life and the rare occurrence of throat diphtheria (while diphtheria of nose and skin occur frequently) can be explained by the absence of these organs at the beginning of life. Accordingly the development of the lymph apparatus on the appendix is minimal, and this could serve as explanation for the rare occurrence of appendicitis in infants.

The following table shows the changes in the blood picture in the course of the first year of life:

Differential Blood Picture

Age	*Erythrocytes (Millions)*	*Hemoglobin*	*Leukocytes*	*Polynuclears*	*Lymphocytes*	*Large Mononuclears*	*Eosinophyls*
Newborn	5.5–6.5*	100–140% 18–22gm	20000– 30000	70%	17%	11%	2%
10 days	5.0–5.5	80–90% 17gm	12000– 15000	37%	46%	16%	1%
6 months	4.0–4.5	70–80% 10–12.5gm	8000– 12000	25%	60%	12%	3%

* Normoblasts up to 10% of every 100 leukocytes are normal. Reticulocytes in the peripheral blood are increased. The hematocrit value is only 35%.

In the blood serum regular changes in the distribution of protein fractions could be observed. Their behaviour toward protein precipitating substances and their stability change from month to month. Only toward the end of the first year does the serum of the infant react like that of the older child. At this time we should also mention the delay of erythrocyte sedimentation rate which reaches the figures of the older child only after the third month. The delay in the formation of immune bodies (see Chapter on Immunity) must also be mentioned as developing function of the first year of life.

The liver, which at time of birth is very large, undergoes a relative decrease in size already during the first year. Of the other inner organs the stomach changes its position, which is almost horizontal in the infant during the first months of life, to a more vertical one. The pylorus is then no longer, as at the early age of the infant, palpable to the right of the middle line, at times under the liver, but almost in the midline. This condition is occasionally of significance in the diagnosis of the hypertrophic pyloric stenosis. The renal cortex grows more intensively than the medulla, not as a result of an increase of glomeruli but through increase of interstitial tissue. The adrenals lose relatively in weight. The thymus still gains in weight during the first year of life. The sweat glands as well as the musculature follow an individually varied tempo in their development. The brain not only doubles its weight during the first nine months but from the relatively little differentiated mass present at birth develops the complicated structure of the central nervous system which is the basis for the great motoric and sensory achievements which the infant acquires up to the end of the first year. At the age of three months the visual neurons are developed. The pyramidal tracts seem to essentially complete their maturation soon after the third month. Details for this are only little known. The great individual differences however, are proven by the extraordinary variation among children in sequence and speed of appearance of the motoric, sensory and emotional achievements.

MENTAL DEVELOPMENT

The advance in physical development is paralleled in the healthy infant by continuous acquisitions of mental abilities. The observation of the development of mental achievements and mental reactions is just as essential for judging the state of health of an infant as the observation of his physical development. Any retardation in the development and in the functions of the body is mirrored in the mental development which is then brought to a stop or even to a regression. Any pathological psychic behaviour in an infant is just as valuable for the physician as a diagnostic symptom as any physical sign pointing toward a disturbance in development. A presentation of the most important data and stages in mental development belong, therefore, into the frame of a nutritional study, since disturbances in the nutritional processes bring about already in the infant striking and partly characteristic changes in behaviour. We might just point out the anxiety of the child suffering from scurvy or the psychic changes, sometimes reaching the stage of severe disturbance of the sensorium, as observed in the course of any acute or chronic diarrhea.

At no period of life is the abundance of newly-learned and newly-acquired knowledge concentrated in such a brief space as in the course of the first year of life. The newborn is essentially an instinctive being whose functions consist of crying, sleeping, sucking and swallowing. In addition, the newborn possesses a few flight and protective reflexes. In the course of the first year the child learns through self-training such accomplishments as grasping, sitting, later standing and finally walking, as well as the first beginnings of speech. The child acquires these accomplishments through constant exercise to which it is automatically driven. In the majority of children these functions appear at about the following ages:

Second month: Raising of the head, either from prone or supine position, corresponding to the full development of the pyramidal tract and the development of restraining influences of the higher centers—smiling after pleasant external stimuli.

Second to third month: Brief fixation of an object.

Third month: Interest in the surroundings.

Second quarter: Balancing of head in erect position; occasionally unsupported sitting with straight back: intentional reaching.

Third quarter: Standing erect; purposeful moving of the body in lying-down position; holding of objects.

Fourth quarter: First attempts at walking; crawling; rarely unaided walking. To this must be remarked that individual variations in this field are especially broad. Individual traits of development, hereditary and educational influences are very strongly felt.

The beginning of motor abilities is often regarded as a result of strengthening and development of the "weak body musculature of the newborn." The hoped for results of infant gymnastics are to accelerate the appearance of these processes. In the final analysis, however, the acquisition of physical abilities depends only on the progress of cerebral development and maturation of the child.

Let us mention some examples for the above: the raising of the head in prone position in the young infant is no measure for the development of its musculature. It is rather a reflex action which is also found in sick infants and those with weak muscles. The appearance of the reflexes in a certain position depends on developmental processes in the central nervous system. Thus the raising of the head in abdominal position is no sign of an unusually well developed musculature. This action takes place at the time when inhibitions, which had hitherto suppressed this function, are removed through the development of higher centers and nerve tracts. It has been observed that children in the first months of life, if supported under the arms, will make typical walking motions with their legs. These motions are motor accomplishments of lower centers which disappear again in the second or third month. Pressure on the sole of a child lying on the stomach will bring about crawling motions in the first months of life. In idiotic children the abilities to sit, stand or run will develop late or not at all, because despite a well developed musculature the maturation of the central nervous system which is necessary for these achievements is lacking. The strength which is displayed already by the youngest infants can be judged by the difficulties encountered in many children in opening the mouth for a view of the throat; or by observing how young children with strongly bent arms can be lifted up, an achievement in which the adult can hardly compete with equal perseverance. If there are other accomplishments which cannot be carried out by the infant it is not due to lack of necessary strength but lack of ability of muscle coordination necessary for movement; this muscle coordination appears only later with progressive development of nerve centers and nerve paths. The ability to move certain muscles and smaller muscle groups separately seems to be of special significance.

The infant during the first months of life reacts to any stimulation with mass movement of the musculature. Uncovering leads to tonic stretching of all four extremities. In all motions both arms and both legs are always moved simultaneously and held symmetrically. Any sign of asymmetry in the motions of an infant is, therefore, of importance to the physician as indication of a disturbance of some kind, either in the lesser moving limb or in the adjacent part of the body or in the nerve centers. The unrestrained urge for motion gradually develops, purely reflexively, into coordinated movements corresponding to the progressing maturation of the nervous system. All these motions appear at first as partial steps toward the final motion after which the child strives, e.g., the aim to walk erect. In the subconscious urge upwards, therefore, every one of these reflexes (labyrinth reflex, positional reflexes, reflexes of neck or limb musculature) is, therefore, constantly and forcibly exercised until the time when the development of higher nerve centers or partly the maturation of inhibitory centers or paths enable the child to carry out complicated coordinated movements and finally to walk erect. Thus one can see children in the fourth to ninth month spending much time lying on the side, the lower arm bent, the upper arm pointed straight upward; if the child is turned to the other side he will immediately assume the same corresponding position of the arms. Other children will partly sit up, with arms supporting the back, without tiring. Once the development of the nervous system is completed these primitive reflexes, although important for the acquisition of positional movements, will disappear. They are suppressed and reappear only occasionally during illness or during disturbances of the higher nerve centers.

Progress in coordination can be observed very nicely in the process of grasping. The grasping reflex can easily be examined and is of practical value in determining the state of mental development of the infant. Already in the first weeks of life the infant responds to a stimulation of the palm of his hand by closing the fingers. Reflexively they approach the object. The thumb does not participate in this process but usually remains vertical next to the proximal phalanges of the other four bent fingers. This grasping corresponds to that of manlike apes, the Primates. A few months later the infant grasps an object which stimulates the palm of his hand with the second to the fifth finger; the thumb does not participate in the motion but rather straightens out in abduction. Only around the age of six months we find an opposition of the thumb which from then on participates actively in the process of grasping. With every attempt at grasping the thumb now persistently strives to place itself in opposition, regardless of its position at the moment of the stimulation of the palm. Disturbances and longer delays in this coordinated mechanism indicate a damage in the cerebral development of the child either through congenital malformation or through acute or chronic illness.

The development of expressive motions goes parallel to the development of the static functions. The newborn and the infant in the first weeks of life can cry and smile. He makes defensive movements with the head and has characteristic facial expressions to stimuli of sour, bitter and sweet tastes. All infants possess certain reflex motions: light causes a closing of the eye lids, pain causes crying, cold causes motions of defense and crying. Smelling and hearing seem to react little to adequate stimulation in the newborn. Only in the second half of the first year the sensory perception is accompanied by appropriate physical movement, indicating that the sensory perception has entered consciousness. This includes the ability to fix the eyes upon an object which is held before the head and seen, the active seeing and the conscious searching for sensory impressions, as in seeing and hearing. The appearance of certain motions in the direction of the stimulus proves the awakened understanding for the stimulation. The appearance of such purposeful motions in response to sensory impressions signifies the first act of recollection of the child. The first complicated psychic emotions such as expressions of fear or conscious affection which were absent in the young infant appear toward the end of the first year of life.

The entire psychic attitude of the young infant toward his surroundings is indifferent. Only around the second quarter of the first year develops a state of comfort and satisfaction which is characteristic for the healthy infant and the absence of which is an early sign of beginning disease. In this sense the majority of acute and chronic nutritional and infectious disturbances affect the mood of the child, causing resigned depression and displeasure. Painful excitement is to some extent characteristic for cases of severe gastro-enteritis and other acute intestinal diseases. Pain and discomfort are evident in children suffering from pylorospasms and some forms of dysentery. Children during the acute phase of scurvy or suffering from severe rickets also show signs of dissatisfaction.

A consideration of the psychic attitude which reflects itself in the facial expression of the child, in the manner of his crying and in his behaviour, may be of significance in the diagnosis and prognosis of some nutritional disturbances. The physician, on the basis of his knowledge of the normal psychic equilibrium of the child, should not neglect from a diagnostic viewpoint the observation of the psychic attitude of his young patient as an essential sign of health and disease also in the field of nutritional disturbances in the infant.

To the above observations should be added the child's desire and pleasure of eating. The presence of these qualities is, with few exceptions (e.g., celiac disease) a sign of the healthy infant. Their disappearance is often an early sign of an incipient disturbance of the nutritional processes. Loss of appetite for any reason whatever is, in the seriously ill child, often the first sign of approaching collapse. On the other hand, as long as a seriously ill infant will accept the food which is offered to him the prognosis of the disease must not be unfavorable.

IMMUNITY OF THE INFANT

The ability to overcome an infection rapidly and without complications is perhaps the best proof for the health of an infant. The infant is immune toward a number of specific infections; this prevents the infection from spreading into actual disease. With the assumption that antibodies pass through the placenta, that is, that the infant has a certain store of immune bodies we can explain the immunity of an infant against measles during the first two to three months of life, assuming, however, that the mother had at one time had measles and that therefore her blood contained antibodies which during pregnancy passed over to the child

While in many animals such as calves, lambs or colts, the antibodies are transmitted through the mother's milk and especially through the colostrum, the transmission of protective substances in rodents and probably also in the human being takes place primarily through the placenta. It is, however, not certain whether this constitutes a transmission of specific antibodies or whether they are merely antigenic substances which, biologically speaking, are immature and non-specific. The anatomical structure of the placenta permits this diaplacental transfer of antibodies in rodents and in the human being. In these species the circulations of mother and infant are separated by a single layer of cells only, while in ruminants several layers of cells separate the organism of the mother from that of the fetus.

It is doubtful whether this assumption is correct and whether it applies generally to other infectious diseases which do not occur in the earliest period of life. The presence of a supply of antibodies is assumed in view of the rare occurrence of measles and diphtheria. In other instances a preceding sensitization through earlier and repeated contact with the organisms is necessary (e.g., scarlet fever, lobar pneumonia), before a renewed contact with the same organisms will lead to the typical disease. On the other hand the organism of the infant is very susceptible to other infections and his reactions more severe and more generalized than in the older child and in the adult under similar nutritional conditions. An example for this is tuberculosis.

The condition which permits the infant to fight off an infection without clinical symptoms or to overcome it by himself quickly and without complications is referred to as natural immunity.

In an attempt to understand the biological action in the process which we call natural immunity we must distinguish two concepts. Firstly the susceptibility for the infection, that is, the number of perceivable diseases which a child suffers in a certain period of life: an individual is never, rarely or frequently sick. Secondly the resistance. Its expression is the manner of defense against the infection—the infection takes a subclinical, light, serious or lethal course. As daily experience shows, susceptibility and resistance run in no way parallel. The degree of susceptibility depends on the condition of the first line of defense which fights off infection. The first line of defense is the condition of the portals through which the infection enters the body. Those are essentially the skin and the mucous membranes of the throat, the air passages, the gastro-intestinal and the urinary tracts, etc. The second line of defense, which determines the resistance, is a combination of the forces which ward off infection, namely, the blood, the phagocytes, the reticulo-endothelial and the lymphoid systems.

The condition for the above is that the organism is basically ready to react to the penetrating germs.

These two different factors, susceptibility and resistance, determine the immunity of the healthy and sick infant. The "tender organism" of the infant is not as lacking in resistance as is widely assumed. On the contrary, it shows a remarkably high degree of immunity toward some infections. For instance the infant in the first months of life has a strikingly low susceptibility to diseases.

This susceptibility is also conditioned by the surroundings. It is lower under good social conditions and greater in narrow crowded quarters. In those countries and those seasons in which it is possible to spend much time in fresh air the number of diseases is smaller than under conditions when the larger part of the year must be spent indoors. To this should also be mentioned the rare occurrence of serious forms of grippal infection during the first months of life. Within a family which is little exposed it is expected that the infant will suffer two to three grippal infections (cold, pharyngitis) in the course of the first year of life. These infections occur for the first time usually in the second half of the first year. Their course is light and without complications. However, this is true only for infants whose development is normal and who can be regarded as healthy. The amount of resistance, however, shows great variations from one individual to the other, and even within the same child. The majority of infants will usually overcome one of the ordinary infections very rapidly. A few children will need more time to recuperate, and one child or another might, despite all therapy, succumb to the same infection which his neighbor will overcome in 2 to 3 days. These different effects of the same infection are not accidental but are determined by a number of factors: 1) constitution; 2) season; 3) age; 4) nutritional state; 5) type of food, and 6) toxicity of the infection.

The constitution is of prime importance in determining the strength of the immune biologic forces of resistance. We find—conditioned by hereditary factors—good and bad producers of antibodies. Thus we see occasionally, although rarely, that apparently healthy children suffer severely from their first infection, resulting in long illness or possibly even death. Usually, however, there have been signs during earlier diseases which indicated the presence of an abnormal body constitution. Difficulties in the raising of the child and a bad nutritional state were the first signs of this special constitution. Only in prematurely born infants can this lack of resistance towards the first infection appear without warning, if after a period of satisfactory development suddenly in the third to sixth month of life a harmless cold may develop into pneumonia and end in death. But apart from this group which is stigmatized from birth, there are a few individuals, especially among children with so-called exudative and lymphatic diathesis, who show a constitutional lack of resistance against any infection. An example of the significance of the constitution for the amount of resistance against infection are mongoloid idiots whose lack of ability to ward off infection is one of the characteristics of the disease.

The season of the year is of great significance for the resistance of the infant. Careful analyses have shown that the number of catarrhal infections is approximately equal in summer and winter. The "Index Infectiosus" is not influenced by seasonal changes. The course of the disease, however, is different in the two seasons. Year after year it has been observed in infant institutions and in homes that in the northern hemisphere from November until spring the nutritional successes are threatened by frequent infections often complicated by secondary infections. In the southern hemisphere where the seasons are reversed, the frequent occurrence of certain grippal infections falls into the months from July to September.

The age of the infant which, as mentioned above, influences the frequency of disease so strongly is also of significance with regard to resistance. Again it is not the youngest and most "delicate" individual who offers the lowest resistance to such diseases but children around and after the middle of the first year of life. Many infections such as chickenpox, diphtheria etc. seem to follow a much milder course with less fever in the first trimester than at a later age. This observation can also be made if smallpox vaccination is made during the first three months of life. If smallpox vaccination is performed at this early age, in contrast to vaccination at the end of the first year of life, it is usually not accompanied by temperature or produces only very low fever

The nutritional state of the infant is decisive for his amount of resistance. Only the truly healthy child has a maximal ability for fighting off disease. Even minor changes in the nutritional state can lead to defeat in the fight against unavoidable infections. The significance of this lowering of resistance will be taken up in detail in the discussion of the nutritional state. In any case, a decline in the nutritional state can often be the essential factor for the lethal outcome of an acute infection. The best prophylaxis against serious infections during infancy is adequate nutrition and the maintenance of a satisfactory nutritional state. We therefore see that most infections take a light course in the eutrophic child. Their course will become the more serious and subject to more complications the more the infant's condition is removed from eutrophia (see p. 181 and Fig. 8).

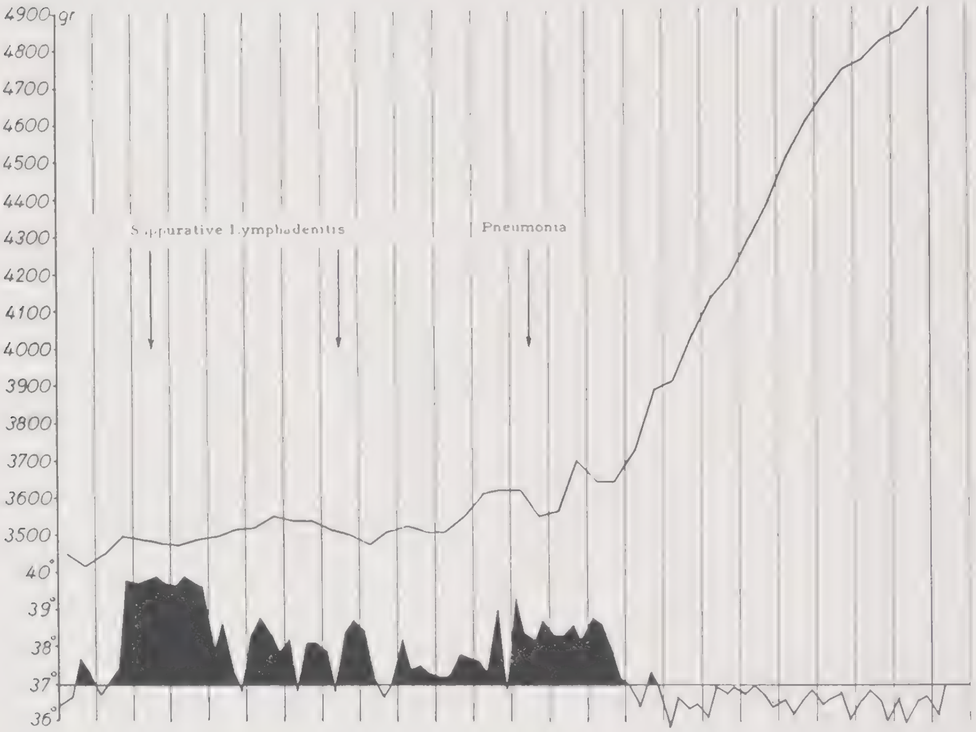

Fig. 8. Infant B. Ch. four months old. 3450 g. Dystrophy. Dysergia in dystrophy. Disappearance of "susceptibility" with increase in weight.

Adequate nutrition is the basis for optimal immunity. Feeding mixtures containing all food components in approximately the same correlation as breast milk build up a high degree of immunity in the infant. Any deviation from this natural correlation, such as one-sided increase of carbohydrates, decrease of fats or insufficient intake of proteins or vitamins has an unfavorable effect on the maintenance of immunity. Any deterioration of the nutritional state is accompanied by hypoproteinemia which in diseases such as the celiac syndrome or lipoid nephrosis may lead to the loss of immunity which is characteristic for these conditions. Hereby the loss of total protein is less important than the decrease of the globulin fraction, especially of the γ globulin which is linked to the immune body formation or may even be identical with it. It must be taken into consideration that an approximately normal globulin content as found by the customary determinations of protein fractions can constitute mainly quantities of α and β globulin fractions and thus give an erroneous picture. Only specific examinations can prove the decline of γ globulin. For the formation of γ globulin in the organism the presence of a number of amino-acids are required such as lysin, methionin, tryptophan and others which are found in complete proteins such as animal protein. Only the presence of all these amino-acids will guarantee adequate antibody formation. Experience shows that also the fat component of the food is of special nutritional value in this respect.

Regular and adequate intake of vitamins is also of significance for the immunity. Total or partial lack of certain vitamins (B and C) leads to a decline in immunity. But even the seemingly definite proofs for the significance of adequate vitamin C intake for the maintenance of normal immunity have not remained without contradictions. Apparently the effect of vitamins on the processes of immunity is only indirect. It is the decline of the nutritional state, connected with any vitamin deficiency, and not the lack of vitamin C in itself which causes the frequent and serious infections as seen for instance in children with prescurvy or scurvy. It seems important to emphasize this fact in order to correct the assumption that it should be possible to improve natural immunity by increasing the vitamin intake, especially vitamins C or B, beyond the normal requirements. This applies also to vitamin A which has, not quite correctly, been termed "anti-infectious fac-

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tor." Excessive intake of vitamins A or C does not result in an increase of natural immunity. There is no proof that vitamins B or D have any direct or indirect influence on the natural immunity of the human organism. The loss of appetite which is connected with any lack in vitamin intake (A, B, C) and the resulting feeding difficulties followed by a decline in the nutritional state are probably the processes which in cases of hypo- and avitaminosis bring about a decline in immunity (susceptibility, loss of normal resistance—dysergia). In the last analysis it is the toxicity of the infection which decides upon its course.

CHAPTER II

Physiology of Nutrition of the Healthy Infant

MILK

The food which provides the infant with the material for building up and maintaining his body is, contrary to the food of the adult, monotonous. During long periods of most intensive development one single food item, milk, covers all nutritive requirements. From this we must conclude that milk contains all the food components necessary for the development of the child in quantitatively and qualitatively sufficient amounts. Protein, fat, carbohydrates, various salts, water and vitamins are the indispensable substances for the undisturbed development of the child. Breast milk no doubt fulfills these requirements. However, the suitability of hetero-specific milk as infant food might be questioned since this milk is determined for the growth of another being—cow, goat, etc.—which develops on different principles and at different speeds. Breast milk can be substituted for infants above all by cow's milk, and also by goat's milk. In some countries donkey's milk and mare's milk are used as substitutes for natural infant food.

The composition of milk of any animal species is practically constant. Quantity and quality of the nutritional substances which form the milk of a species vary within very narrow limits. Even in the course of long periods of lactation no essential changes can be found except during the first days of lactation. The protein, which is the most valuable and irreplaceable substance for the growth process shows the least variations. All other nutritive substances show somewhat greater variations, depending on external factors, particularly the nutrition of the mother. Breast milk shows the following differences in the various stages of lactation:

	Fat (*g%*)	*Sugar* (*g%*)	*Protein* (*g%*)	*Ash* (*g%*)
Colostrum	2.83	7.59	2.25	0.31
Transitional Milk	4.39	7.79	1.56	0.24
Mature Milk	3.26	7.50	1.15	0.21
Late Milk	3.16	7.47	1.07	0.20

In comparing the milk of different species far-reaching differences are found as to quantity and partly also quality of the various nutritive substances. The causes for the varied successes in natural and artificial feeding of the human infant were occasionally attributed to these differences. The divergent results of infant feeding with breast milk on the one hand and with heterospecific milk (cow's milk, goat's milk) on the other hand were explained at times by the differences in the amount of protein, the varying fat or sugar contents or the great differences in salt contents.

The amounts of protein and salts show considerable variations in the different types of milk.

Species	*Period for Doubling Birth Weight* (*Days*)	*Protein* (*g%*)	*Salt* (*g%*)	*Calcium* (*g%*)	*Phosphorus* (*g%*)
Human being	180	1.6	0.2	0.033	0.047
Horse	60	2.0	0.4	0.124	0.131
Cow	47	3.5	0.7	0.160	0.197
Goat	22	3.7	0.8	0.197	0.284
Sheep	15	4.9	0.8	0.245	0.293
Pig	14	5.2	0.8	0.249	0.308
Cat	9.5	7.0	1.0	—	—
Rabbit	6	10.4	2.5	0.891	0.997

These great differences are not accidental but are the expression of a generally applicable biological principle: the greater the intensity of growth of a certain species (measured by the time required for doubling the birth weight) the higher the protein content of the milk. A similar dependency on the speed of growth of a type of animal is shown also in the amounts of salts contained in the milk. They as well as calcium and phosphates are, next to the protein, essential for the formation of new tissue. For the practical application of feeding the human infant, it is apparent that a diet of pure cow's or goat's milk, in the same quantities as the child takes from his mother's breast and sufficient to cover his satiation, brings about, apart from all other differences, above all an excessive protein intake. Too strong a dilution of milk, on the other hand, may lead to a protein deficiency in view of the essential function of this most important formative material.

During various periods of pediatric research protein has again and again been considered the cause for the less successful results of artificial feeding. The theory of the "difficulties of digesting cow's milk protein," resulting in "harmful food remnants in the intestines" was superseded by the assumption that the biologically important qualities of breast milk protein were the cause for the superiority of homologous protein. It was presumed that the assimilation of nitrogen containing substances could proceed without difficulties and with the least effort only in the form of breast milk protein. These views, however, do not agree with our knowledge of protein digestion in the intestines. Against this theory of the difficult assimilation of cow's milk protein the objection seems rightful that all protein substances, even those contained in breast milk, must be broken down into polypeptides and amino-acids before their entrance into circulation and the building of tissues. However, it has been proven that the various amino-acids or amino-acid complexes are of varying value in the building up of the tissues. One could find the explanation for the inferiority of cow's milk as compared to breast milk in the nutri-

AMINO-ACIDS OF COW'S MILK IN PERCENTAGES OF TOTAL PROTEIN

Essential Amino-acids:	*% of Protein*	*Non-essential Amino-acids:*	*% of Protein*
Lysine	6.0	Glycine	0.5
Tryptophane	2.2	Alanine	1.9
Histidine	2.5	Aspartic acid	1.8
Phenylalanine	3.9	Serine	0.5
Leucine	9.7	Glutamic acid	20.0
Isoleucine	1.4	Hydroglutamic acid	10.0
Methionine	0.4	Proline	8.0
Valine	7.9	Hydroxyproline	0.3
Arginine	4.0	Cystine	0.4
		Tyrosine	6.0
	±40.0		±50.0

tion of infants in the lack of some of these amino-acids. Vital or essential amino-acids are those which cannot be formed in the metabolism of the human organism. Therefore infants are dependent on their intake with the food.

The essential amino-acids are contained in largest quantities in the albumin part of milk protein. The albumin content of the milk can, therefore, be regarded as approximate guide for judging the value of the protein.

AMINO-ACIDS OF CASEINOGEN AND ALBUMIN

	Caseinogen	*Lactalbumin*
Cystine—N	1.30%	2.15%
Lysine—N	9.46%	12.54%
Arginine—N	9.31%	7.56%
Histidine—N	6.55%	4.44%

The albumin content is higher in breast milk than in cow's milk:

100g cow's milk contain	0.2–0.3g	Albumin,	2.7–3.0g	Casein
100g breast milk contain	0.6g	Albumin,	0.98g	Casein

From the above figures it would be correct to consider breast milk as albumin-milk and cow's milk as casein-milk.

This seemed to prove the lower value of cow's milk protein for the development of the child. However, it must be considered that the albumin deficiency, even in the customary dilutions of cow's milk, hardly ever reaches the degree which would lead to arrest of development in animal experiments as a result of complete absence of certain essential amino-acids. It would be more likely to consider relationships of vital amino-acids to the formation of fermentative secretions or of purine bodies which might perhaps be of significance for the development of a good immunity of the infant.

For biological problems it is not without interest to know that the albumin and globulin in the whey are in each case identical with the albumin and globulin of the blood serum of the same species. Finally one would have to bear in mind the behaviour of the albuminous substances of the milk, characteristic for each species, towards influences which cause coagulation of the milk. Thus, for instance, it is much more difficult to bring about curdling of breast milk through lab ferment, acids or salts than cow's milk. The resulting coagulation is again type-specific: breast milk causes fine flocculation, cow's milk, under the same conditions, large curds. Apart from the type of protein, however, the salt and acid contents and the different buffer actions of the various kinds of milk are of decisive significance for the type of resulting protein coagulation.

Recently there seems to be almost a return to the old concepts. Again the difficult digestibility of cow's milk casein moves to the foreground. The higher casein content of cow's milk necessitates a longer stay in the stomach. This delay in the emptying time of the stomach, it is thought, spreads to the entire intestinal tract and thus causes the beginnings of a disturbance of motility, a stagnation of the intestinal contents and thus the first basis for the development of a nutritional disturbance. Peptic predigestion of cow's milk casein which shortens its stay in the stomach is supposedly one way which should make possible an adjustment of artificial feeding to natural feeding (see chapter on Digestion). The good results obtained by adding amino-acids to the diet of particularly delicate prematures could be regarded as support of this theory.

But even the theories on the significance of the specificity or hetero-specificity of milk protein are revived in a somewhat altered form. The development of the infant depends, therefore, on the transfer of the "living protein substances of breast milk" to the child. This includes immune bodies, antitoxins and ferments. In animal experiments it could, in fact, be proven in the young that the transfer of immune bodies takes place only with type-specific milk. Also the high content of certain antitoxins in the serum which, as in diphtheria, can be demonstrated directly in the test tube or by the Schick test, is further proof of this fact. The transferred immunity against certain infectious diseases which should last longer in the breast fed child than in the artificially fed child, in fact longer in the infant nursed by his own mother than in that raised by a wet nurse, supports the theory that the specificity of milk is of importance for the development of the infant. These explanations, however, are hardly sufficient to explain the less successful results of raising human infants on animal milk.

The so-called whey exchange experiments also speak against the significance of protein as the essential cause for the more unfavorable nutritional successes with artificial feeding. Not the protein but the type of whey in which the protein was given is important for the development of the child. Casein of cow's milk in breast milk whey is preferable to breast milk casein in cow's milk whey (see below).

On the whole all attempts to attribute the cause for the different results of breast feeding and artificial feeding to the milk proteins have so far not shown any clear results. On the basis of the results of the whey exchange experiments the whey of the milk was regarded as cause for the different results obtained with cow's milk and breast milk feeding. The considerable absolute differences in salt content of the two types of milk seemed to support the justification for such a theory.

MINERAL SUBSTANCES IN DIFFERENT TYPES OF MILK
(in mg %)

	Na	*K*	*Ca*	*Mg*	*Cl*	*P*	*Fe*
Breast milk	14	53	30	3.6	30	25	0.15
Cow's milk	15	160	136	12	97	98	0.18
Goat's milk	79	145	128	12	100	100	0.21

Essential also are the quantities of anorganic and organic phosphorus as contained in breast milk and cow's milk:

Breast milk: 5–6 mg% anorganic, 6.3 mg% organic phosphorus
Cow's milk: 60 mg% anorganic, 15 mg% organic phosphorus

The relationship of Na, K and Cl, the salts which are important for water metabolism and acid-base balance, is breast milk: cow's milk—1:3. Cow's milk contains more than double the amount of salts found in breast milk (72 mg% against 30 mg%). The differences remain even if one considers that the customary dilutions of cow's milk ($^{1}/_{2}$ or even $^{1}/_{3}$ milk) somewhat equalize the amounts.

In addition, minimal quantities of iodine, fluorine, magnesium, copper and zinc are present.

COMPARISON OF SALTS IN BREAST MILK WITH $1/2$ AND $1/3$ COW'S MILK

	g in 100 kg Breast Milk	*g in 33.3 kg Cow's Milk ($1/3$ Milk)*	*g in 50 kg Cow's Milk ($1/2$ Milk)*	*g in 100 kg Cow's Milk (Whole Milk)*
Potassium K_2O	69.0	63.0	95.0	188.5
Calcium CaO	42.0	57.0	86.0	172.0
Sodium Na_2O	16.0	15.0	23.0	46.5
Magnesium MgO	6.8	6.8	10.2	20.5
Alkalis	133.8	141.8	214.2	427.5
Chlorine Cl	29.4	27.0	41.0	82.0
Phosphoric acid P_2O_5	33.4	75.0	113.0	225.1
	62.8	102.0	154.0	307.1

A series of observations speaks for a certain significance of type-specific whey. The nature of this superiority can, however, not yet be determined exactly. The following can be listed in this respect: 1) The better cell respiration in the medium of breast milk whey, as measured by the oxygen consumption of surviving cells; 2) the better cell work as measured by the ability of the cells to resorb; 3) the evidence of better work of the gastric and intestinal ferments in human milk whey, and 4) the better resorption of lactose from segments of the intestine of young goats in the presence of isospecific whey.

However, the absolute values of the different salts are not decisive. More important is the correlation of the individual salts for the biologic processes, since the individual salts influence each other at times in a retarding, at times in a promoting manner. It is important to note, therefore, that despite the considerable absolute differences, the correlation of the salts of breast milk and cow's milk is very similar.

	g in 100 kg Breast Milk	%	*g in 100 kg Cow's Milk*	%
Potassium K_2O	69.0	52	188.5	44
Calcium CaO	42.0	31	172.0	40
Sodium Na_2O	16.0	12	46.5	11
Magnesium MgO	6.8	5	20.5	5
	133.8	100	427.5	100

But even under the assumption, supported by clinical and experimental observations, that all vital processes proceed more favorably in the surroundings of isospecific breast milk whey than in cow's milk whey, a final decisive proof for the biologic significance of the difference in salts in cow's milk or breast milk respectively has not yet been established.

Differences of carbohydrates and milk fats also furnish no explanation for the different results of artificial and natural feeding. There are no qualitative differences in the sugar content of cow's milk and breast milk. In both types of milk as well as in goat's milk the carbohydrate is present in form of lactose. The quantitative differences between the types of milk are, however, considerable. Their relationship to the quantity of protein is inverse, that is, the higher the protein content of the milk the lower ordinarily the content in sugar.

	Protein	*Lactose*	*Fat*	*Ash*	*Calories*
Breast milk	1.6 g/100cc	7.0 g/100cc	3.6 g/100cc	0.4 g/100cc	700/liter
Cow's milk	3.4 "	3.8 "	3.4 "	0.7 "	670/liter
Goat's milk	3.7 "	4.0 "	4.3 "		
Sheep's milk	4.7 "	4.6 "	5.0 "		
Donkey's milk	2.1 "	6.4 "	1.5 "		

An equalization of the considerable differences in various types of milk with reference to their sugar content is easily possible and is accomplished in artificial feeding by corresponding additions. It is interesting to note that the quantities of lactose which in a breast fed child are easily digested and assimilated, not infrequently lead to nutritional disturbances if offered in chemically similar combinations of cow's milk mixtures (e.g., ½ milk + 7% sugar).

What applies to the carbohydrates is also true for the fats. Despite some chemical and physical differences which exist between breast milk and cow's milk—cow's milk is rich in low molecular fatty acids (about 20%) which are almost completely lacking in breast milk (2%)—these variations are not sufficient to explain the differences in results in natural and artificial feeding.

	Breast Milk	*Cow's Milk*
Fat content	4%	1.5–4.5% (3.2%)
Melting point	30–34°	31–36°
Solidification point	20°	19–24°
Specific gravity	0.966	0.9307
Iodine value	45	30–40
Reichel-Meissl's number (volatile fatty acids)	2.5	25–27
Linoleic acid*	7%	Traces–0

° Linoleic acid is the most important unsaturated fatty acid for the nutrition of the infant.

From a physico-chemical point of view the fat contained in breast milk is not altogether similar to that of cow's milk. The emulsion of breast milk fat is finer than that of cow's milk fat. The cholesterol content differs in the two types of milk, although it varies depending on nutrition and stage of lactation.

CHOLESTEROL CONTENT OF MILK

Breast milk contains about 12.5–19.2 mg in 1000 cc. milk
Cow's milk contains about 12.5–15.1 mg in 1000 cc. milk
or: 100g breast milk fat contain 0.427–0.515g Cholesterol
100g cow's milk fat contain 0.343–0.364g Cholesterol

The differences in the physico-chemical behaviour of the two types of milk also do not explain the differences in feeding results of breast milk or cow's milk. Fresh cow's milk with a pH of 6.4 to 6.8 is more acid than breast milk with a pH of 7.0 to 7.2.

Finally even the amount of ferments present in the milk does not contribute to the explanation for a better tolerance of breast milk.

	Breast Milk	*Cow's Milk*
Protease	O	O
Lipase	+	O (?)
Lactase	?	Trace
Peroxydase	Varying	+
Indirect reductase, Methylene blue splitting in the presence of reductase	O	+
Diastase (Amylase)		+

The contents of essential vitamins in breast milk and cow's milk are shown in the following table:

Vitamin:	*A Internatl. Units Per Liter*	*B_1 γ/liter*	*B_2 γ/liter*	*Nicotinic Acid γ/liter*	*B_6 γ/liter*	*C mg%*	*D I.U.*	*E mg%*
Breast milk	150–400	300–600	1600	250–500	—	4–7	minimal (2–3)	3 mg% first days of life, later about 0.1 mg%
Cow's milk	300–750	300–750	2000–3000	1000–6000	100–300	0.5–2.0	minimal	0.06 mg%

These values, however, vary with the nutrition of the milk donor and in cow's milk with the treatment of milk after the milking (boiling, pasteurizing, diluting). The following observations can be made regarding the relationship of the nutrition of the mother and the vitamin content of her milk: only traces of vitamin D are transferred to breast milk. The vitamin C level in breast milk remains unaffected by variations in the vitamin C intake of the mother for a long period of time. Of the B complex, the riboflavin content in the breast milk is influenced by the food of the mother. The thiamine content, on the other hand, remains almost constant throughout the entire period of lactation. But the thiamine content seems to be dependent on the amount of milk production: if the quantity of milk is reduced, the thiamine content also decreases. With the change in fat contents in the course of a meal (increase of fats towards the end of the meal) the amount of vitamin A is also increased. If the nursing mother takes vitamin A, in one dose or for a short period of time, the vitamin A content in her milk also rises temporarily. The practical conclusions of all these above facts are: in order to obtain in breast milk an amount of vitamin A, B and C sufficient for the infant (the amounts of vitamin D cannot be increased) the food intake of the nursing mother must contain vitamins A and C constantly and in sufficient quantities, in form of fruit and vegetables. The vitamin B requirements of the infant are adequately covered if the mother receives an average normal diet.

None of the as yet tangible differences between cow's milk and breast milk—and this applies also to other types of milk which are occasionally used for infant feeding—can explain the different feeding results of the two kinds of milk in the practice of infant nutrition. In recent times research has therefore turned away—possibly wrongly—from the study of milk as chemical and colloidal compound. The turning away from this altogether difficult but unsuccessful research work was bound to occur since experience shows that the development of the child can be accomplished also with feeding mixtures and milk preparations which chemically and physically do not show much resemblance to breast milk. Only one condition must be fulfilled, namely, that all food components must be represented in adequate quantity and in good quality. Efforts are therefore directed today towards regulations for the production of a grade A milk from which a usable artificial infant food can be prepared following simple empirically found rules.

Breast milk can practically always be considered as perfect, assuming that the nutrition of the mother is not seriously defective. The fact that infants whose mothers are afflicted with Beri Beri due to vitamin B_1 deficiency suffer from the same disease shows that even the milk gland can supply adequate secretion only if all elements are furnished by the blood stream. Only one substance is lacking in breast milk in adequate quantities: mother's milk has a relatively low or almost absent supply of anti-rachitic vitamin. This is the reason why even the nursing infant is subject to rickets unless he is given vitamin D by some other route. Irradiation of the mother or supplementation of the diet by known vitamin carriers (butter, cod liver oil) cannot increase the content of this vitamin in breast milk. These measures are not sufficient to dispense with anti-rachitic prophylaxis even in the breast fed child. Except for the vitamin D deficiency, breast milk is in every respect perfect and adequate for the first five to six months of life of the infant.

The requirements as to what constitutes today a good cow's milk have changed considerably compared to the requirements of about 40 years ago when *Soxhlet* established his system for the preparation of an adequate infant food. Former requirements can be summarized as follows: milk as free of germs as possible through good hygienic conditions of stables, dry feeding of milk cows, rapid transport of milk to the consumer, possibly single or even repeated partial sterilizing processes (pasteurizing etc.); adequate fat content of food, elimination of cows suffering from tuberculosis, Bang's disease (or goats suffering from Malta Fever) and of all cows afflicted with suppurative mastitis. The need for a germ poor milk, particularly the destruction of disease producing germs, is even today of prime importance. The recognition of the significance of vitamins for the health of the child has led to a discontinuation of strict dry feeding methods and to the addition of vitamin carriers to the fodder of milk cows since only in this manner an adequate vitamin C content of the milk can be assured. Pasturing in summer, addition of beets, sloppy food, carefully dried hay (silage) in winter, etc. are today requirements for milk cows supplying milk for infants. The possible uncleanliness of the milk caused by this feeding method must be reduced by careful hygiene of the stables so that the milk obtained from the cow is as free of germs as possible.

DESTRUCTION OF DISEASE GERMS IN THE MILK DURING PASTEURIZATION

	Degrees Celsius	*Time Required for Destruction (in Minutes)*
Tuberculosis	60°	20′
Brucella abortus	60°	15′
Salmonella	60°	5′
Shigella	60°	10′
Streptococci	55–63°	5′
Coli	65°	45–60′

This requirement (pasteurization) is necessary although even the most careful process of freeing the milk of germs by pasteurizing, sterilizing, etc. diminishes or destroys part of the vitamins. Even a single pasteurization process at temperatures between 60 to 70°, has proven harmful with regard to the preservation of vitamins. The vitamin C content of cow's milk decreases after pasteurization from 1 to 2 mg% to half of that amount (0.5 to 1 mg%).

The previously raised doubts against the manufacture and utilization of dried milk preparations for infant nutrition are no longer valid today. Through modern processes the vitamin content of many types of powdered milk is preserved to a great extent. An early supplementation of the diet in infants fed with dried milk, the vitamin content of which is not known, is always necessary. Powered whole milk is, therefore, today rightfully recommended as adequate preparation for the artificial feeding of infants, especially in large cities where the supply of fresh milk to the population offers certain difficulties.

The heating of milk, which is customary in every household, causes a number of changes in the milk which as yet are known only to a small extent. Their significance for digestive processes cannot yet be evaluated. Even a short boiling alters the color of the milk. It no longer looks white but assumes a bluish or yellowish appearance. A large part of the gases which have entered the milk during the milking process, during transport, etc. (mainly carbonic acid) are removed so that boiled milk reacts less sour than raw milk. The changes in the casein are no doubt considerable. The difference in the behaviour of the casein of boiled and unboiled milk toward the lab ferment is characteristic for these changes although it has so far not been possible to analyze the nature of the change in the protein molecule. A transfer of calcium into different compounds is no doubt part of the process. The fat in boiled milk tends to accumulate in larger drops. As a result boiled milk accumulates less cream on the surface than unboiled milk. The customary short heating produces no changes in the carbohydrates. Caramelization of lactose takes place only if milk is heated intensively and for longer periods of time. A short boiling of the milk does not essentially decrease the vitamin content.

For discovering cow's milk admixtures to breast milk the neutral red test according to *Moro* is used: several cc of milk are dissolved with two to three drops of neutral red solution (1%) for a short time (half a minute) in hot water. Breast milk turns yellow or orange. If it turns reddish or red the milk is either contaminated with bacteria or it is mixed with cow's milk. Then the following test is indicated: 5 cc milk are centrifuged, 1 cc pipetted off after separating the milk from the fat layer. 1 cc of this milk, simple molar acetate buffer of pH 4.6 and 2% calcium chloratum siccum are added in a centrifuge tube. To this solution

5 cc distilled water is added at 70° (not warmed up), the tube closed with a rubber stopper, mixed and centrifuged for 40 minutes.

Cow's milk casein accumulates at the tip of the tube (5% admixture can be discovered).

In case of dilution with water a determination of the specific gravity is usually sufficient. Breast milk has a specific gravity of 1030–1032. If water is added the specific gravity decreases.

COLOSTRUM

The milk produced during the first days of lactation differs from the milk which, apart from minor variations in composition, is secreted uniformly through the period of lactation in the human being and the animal. This first milk, the colostrum, can be obtained from the breast glands in small quantities by pressure already during the latter half of pregnancy. The colostrum differs from the milk which is produced later by a thicker, somewhat sticky consistency, a stronger yellowish color and rapid coagulability at relatively low temperature. Microscopically the colostrum shows, apart from fat droplets, roundish formations filled with bright shiny droplets larger than fat droplets. These formations are leukocytes which have phagocyted fat. The formations, which are typical for colostrum, disappear as soon as lactation advances. They can reappear if the breasts are not completely emptied. They are, therefore, occasionally found again at the time of weaning and their reappearance in the milk can point towards an insufficient emptying of the breasts. The leukocytes essentially cause also the high protein content of colostrum in comparison to the milk of later nursing periods.

COMPOSITION OF COLOSTRUM

	Cow's Milk	*Breast Milk*
Fat	3.6%	3.6%
Protein	4.0%	3.7%
Lactose	4.0%	5.1%

This conclusion seems justified since the milk protein content of the colostrum, that is, casein and albumin, does not differ essentially from that of later milk° The lactose content of the colostrum is, however, lower. The fat content apparently shows great variations. In the earliest period of lactation the type of fat which is secreted corresponds in composition to body fat while fat of the quality of full milk fat appears in the milk only with the establishment of milk secretion. The considerably higher ash content of the colostrum is perhaps essential for the processes of the first days of life. Colostrum contains almost double the total amount of ash of mature milk (3.05:1.84). The main increases are in the sodium content (0.53:0.19 Na_2O) and potassium content (0.80:0.53 K_2O). The caloric value of colostrum varies, depending on its greatly fluctuating fat content, between 500–1500 calories per liter. Colostrum is rich in vitamin A (170 to 230 Internatl. Units per 100 cc) and Carotin (40 to 60 Internatl. Units per 100 cc) which produces the yellow color of the colostrum. The vitamin C content of the colostrum is about 7 mg%. Finally in some species the abundance of immune bodies of different types in the colostrum is biologically significant.

METABOLISM OF THE HEALTHY INFANT

The study of metabolic processes was originally stimulated by the desire to find out well defined, primary metabolic disturbances in acute and chronic nutritional illness such as exist in diabetes mellitus. This aim was not accomplished, or accomplished only very recently in a few rare diseases (e.g., aminoaciduria). The investigations did, however, bring an understanding for the observations made on infants suffering from nutritional disturbances and the determinations of the metabolism resulted in important therapeutic progress. The knowledge of peculiarities of metabolism in healthy and sick infants is therefore of great practical importance. From the abundance of material which was gathered at great effort, only those points which are essential from a practical point of view can here be presented. Only on the basis of an understanding of the complicated processes of digestion and metabolism of the infant will the physician be in a position to understand and to treat conditions which

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° Other cells found in colostrum according to Engel are epithelial cells which are shed by the breast glands at the time when it begins to function.

cause changes or disturbances in the normal process of metabolism. The various phases of metabolism (transport of food in the gastro-intestinal tract, digestion, food absorption and assimilation) will be discussed one after the other. It must be considered, however, that all these processes actually take place simultaneously and in closest mutual dependency.

MECHANISM OF FOOD TRANSPORT IN THE GASTRO-INTESTINAL TRACT

Sucking is a motoric function, characteristic for the infant, and is regulated from a nervous center in the medulla. The sucking of the infant is not identical with the inspiratory drawing in or sucking of the older child. By lowering the lower jaw and raising the base of the tongue to the soft palate and by firm, airtight grasping of the sucking object (nipple of breast or bottle) the mouth cavity develops a pressure which is different from the outside air so that milk flows from the milk ducts or from the bottle into the mouth cavity. The difference in pressure in the mouth cavity of 10 to 30 cc of water, created by the sucking, is certainly not sufficient, however, to empty the breast. More important than the initial sucking is the second step of the sucking process, the raising of the lower jaw resulting in pressure on the nipple which in turn causes the expression of the milk. The so-called sucking of the infant resembles actually a chewing motion. In addition there is a third factor, the process of active secretion, performed by the milk gland as by any other glandular organ. Thus in some women a milk flow can be produced even before the infant is put to the breast by the mere desire to nurse the infant.

After a larger quantity of milk has accumulated in the oral cavity through sucking and pressing, a swallowing motion ensues. While the amount of milk which reaches the oral cavity during the first minutes of the feeding is so large that every sucking motion is followed by a swallowing motion, the number of swallowing motions is reduced in the course of the meal. Accordingly, the swallowing motions in bottle feeding are also more frequent in the first minutes of eager and hasty drinking, while towards the end of the meal the child is satisfied and tired and drinks slower and with diminished force. The ability to normally suck and swallow is acquired by some infants only in the course of the first weeks of life. Many children display at first little talent for this task. There is a great number of infants who fail to accomplish the airtight closing of the oral cavity in the first phase of the sucking process and thus swallow large amounts of air in the course of the meal. By raising the infant up after the completion of the meal the swallowed air should be removed from the stomach as far as possible because if the air comes up in a recumbent position it carries along food and thus causes regurgitation and vomiting.

Sucking and swallowing are possible even in decerebrated malformations. However, severe malformations of the facial skull can make it impossible for the infant to drink from the breast or bottle. This does not include the hare lip and cleft palate; children with these malformations usually learn after a short time to drink sufficient quantities from breast or bottle. A greatly receding or underdeveloped lower jaw (micrognathia), a macroglossia or the partial or complete congenital atresia of one or both choanae are occasionally much more disturbing and can make it impossible to carry out the sucking act.

The stomach of the infant is more ball-shaped than that of the adult. It lies almost completely in the left half of the abdomen and only the pylorus extends beyond the middle line to the right. If the stomach is full and the abdominal wall soft, the pylorus is palpable to the right of the spinal column. The lowest point of the empty stomach is the pylorus, and of the full stomach it is a point in the large curvature. The muscles of the gastric wall are moderately developed and also the secreting cells of the different types are not fully matured. For this reason the motoric action of the stomach is little developed in young infants. This causes the incomplete development of the peristolic reflex (ability of the stomach to contract concentrically around its contents and to adjust to its contents) and the not clearly marked separation in the healthy child of the pars pylorica from the fundus.

The stomach capacity is not sufficient for the amount of a normal milk feeding. This applies particularly to the newborn period when the capacity is often as low as 20 to 60 cc. Subsequently the vital capacity increases rapidly but the stomach is never sufficiently large to hold a quantity of 150 to 200g of milk. In consequence, part of the food intake, particularly the separated whey and other fluids, are passed on into the duodenum already during the course of the meal. The largest part of a meal leaves the stomach within twenty minutes. Cheese curds and fat remain in the stomach. Soft food remains in the stomach longer than liquid food. A vegetable meal remains in the stomach for a particularly long time (four to five hours).

The entire food quantity taken in at one meal, which amounts up to 200g and even more leaves, in case of breast milk, the stomach after about two hours. In artificial feeding $2^1/_2$ to 4 hours may pass until the stomach is completely emptied. Delay in the emptying time depends partly on the fat content of the food, partly also on the quantity and quality of the heterospecific protein (casein and whey protein). The process of emptying the stomach is according to all experience very labile in infants. Especially in the artificially fed child delays in these functions occur frequently. The possibility of stagnation of undigested food remnants, loaded with bacteria, before the entrance to the delicate duodenum is frequently the basis for the development of diarrheal diseases. Delay of the emptying time of the stomach in infants occurs mainly by parenteral infections, fever, pathologic spasms of the pylorus and acute diarrheal diseases. Atonia of the stomach is therefore not uncommon in all these conditions. The lability of stomach motility is also demonstrated by the effortless vomiting and regurgitation of infants. Although the musculature of the abdominal wall and the diaphragm has more significance for the motor action of vomiting than the activity of the stomach musculature itself and although vomiting is caused basically through stimulation originating from nervous centers, we find that loss of food through the esophagus is much more frequent in the infant than in the older child or the adult. The obstacles opposing such a wrong course of the food are certainly very insignificant in the young child. This experience is confirmed in case of pylorospasms or pyloric stenosis, where during one peristaltic wave of the stomach in the direction of the pylorus, with the help of abdominal and diaphragmatic muscles the food is expelled through fundus and cardia.

The partially digested food passes through duodenum and small intestines in relatively short time although the distance is rather considerable. Despite the fact that indications as to the length of the intestines vary greatly and are easily subject to errors, the distance from pylorus to rectum can be estimated between 2 to 6 meter (three to 11 times the body length). In the healthy individual transport of food in the intestines is basically only in a caudal direction (polarization of the intestines). Motions in cranial direction are possible; they appear, however, only in pathological conditions such as in regurgitation, vomiting, rumination, vomiting of bile stained or fecal material, etc. The tonus of the intestinal wall is of significance for the transport of food; it progresses wavelike and in case it meets obstacles it is visible in form of violent undulations underneath the abdominal wall. The normal tonus of the musculature depends upon an adequate oxygen supply of the intestines. Anoxemia and stasis involving the intestines which occur in diseases of heart and lungs, in collapse, shock or severe nutritional disturbances, lead to a loss of tonus, to meteorism and finally to atonia and paralytic ileus.

Apart from the rhythmic, pendulous motions which cause the mixing of the food the small intestine executes progressive peristaltic motions so that the partially digested food, liquid or semiliquid, which is pushed down from the stomach, is rapidly processed like on a conveyor belt. This procedure makes it possible to terminate the digestive process of the entire food intake within a short period of time. It also avoids the possibility that the ingested food might stagnate and decompose somewhere along the way. The scarcity, in fact almost complete absence of bacteria in the small intestine (see later) is due essentially to the rapid and constant self-cleansing of these sections of the intestinal tract. In the infant the time required for a meal to travel from the pylorus to the beginning of the large intestine amounts to about 5–8 hours.

The food remnants which reach the large intestine in form of a thin pulp, progress there only very slowly since in addition to the motions which help to mix and transport the food there appear, in contrast to the duodenum and small intestine, anti-peristaltic waves which delay the progress of the intestinal contents. These anti-peristaltic movements of the intestines are of great significance for the stool formation (thickening of the stool) (see later). The passage through the large intestines therefore requires eight to 10 hours, despite the relatively short distance.

The mechanism of stool elimination is apparently not much developed in many young infants. This is shown by the difficulty with which many children, especially breast fed infants, eliminate only after several days a stool which is absolutely not hard and solid but pasty and of normal consistency. It is further shown by the effort which many infants, turning blue and red in the face, have to make in order to expel a stool. The greatest effort is thereby made by the abdominal wall rather than by active and, in the opinion of some mothers, painful labour of the intestines.

The entire motor activity of the intestines is under the control of the nervous system and stimulated indirectly by the influence of the products of digestion upon the intestinal mucosa and nerves. The rhythm of the opening of the pylorus is of decisive influence upon the entire subsequent progress of intestinal motions. Closing of the pylorus causes inhibition of intestinal motor activities. It is furthermore possible that chemical stimuli which reach the nervous plexus of the intestinal wall or stimuli which originate centrally and reach the intestinal tract by way of the autonomous nervous system influence the peristalsis of the small and large intestines to such an extent that they produce atonia, constipation or diarrhea. It cannot always be differentiated which part the smooth musculature and which part the nervous apparatus, the so-called plexus myentericus, have on these processes since muscles and nerves are most likely coordinated at all times.

Influences of external nervous stimuli (extrinsic nerves) are not definitely established. There are great differences in the nervous supply of the various sections of the intestinal tract. They can perhaps be explained by the fact that not those stimuli are decisive which originate directly from the nerves but that the effect on the intestinal wall is created by the intervention of chemical products which are formed in the various sections of the intestines.

DIGESTION

On the long path from mouth to rectum the ingested food is separated into substances necessary and usable for the building up of the organism and into unusable remnants. Two forces contribute to this process: digestive juices and intestinal bacteria.

Digestion by Ferments

Chemical digestion through the ferments of intestinal glands and the glandular appendages of the intestinal tract (salivary glands, liver, pancreas) takes place in the infant from the first day of life, with only few exceptions due to immaturity, through the same secretions which are also effective in the older child and the adult. In the newborn too, digestion starts already in the oral cavity through the influence of the saliva. The quantity of saliva is initially very small but increases already after a short time to considerable amounts. In this respect milk (as liquid food) assumes a special place compared to all other liquids: milk is the only "liquid" which stimulates the salivary glands to large secretions of mucous saliva, so that saliva amounting to 10 to 20% of the food volume is secreted. The reaction of infant saliva is slightly acid in the first period of life. Only with the abundant salivary flow of older infants we see, constantly and independent of external influences, a reaction between pH 7.6 and pH 6.8, i.e., the saliva becomes then a neutral or slightly alkaline fluid with a pH around 7.0. The quantity of saliva decreases considerably in case of illness. The measurable decrease of the secretion corresponds to the clinical finding of dry, lustreless mucous membranes in the mouth of infants suffering from nutritional disturbances or fever. This decrease in saliva is perhaps more significant for the digestive process than the reduction of the diastase content of the saliva which, by the way, does not consistently diminish corresponding to the seriousness of the disease.

Gastric digestion is dependent upon the protein, fat and salt content of the food. As a result, chemical digestive processes of the stomach in case of natural feeding are not in complete accord with those taking place with the use of artificial feeding mixtures. Gastric secretion is lower in naturally than in artificially fed infants. Of the protein splitting ferments the newborn furnishes already pepsin, cathepsin and lab (if this is recognized as a separate ferment) from the gastric mucosa. The action of pepsin, however, is at least in the youngest infants limited or maybe totally lacking since the acidity which is necessary to permit action of the pepsin is not yet reached in the stomach of the healthy infant. The true acidity of the gastric juices corresponds at the height of digestion to a pH of about 4.0 to 5.0, with the acidity increasing with the growth of the child. Pepsin reaches its optimum of efficacy only in a pH of 1.8 to 2.4 so that there is practically no protein digestion through pepsin in the stomach of the infant. Its place is taken by cathepsin which is effective already in less acid surroundings (pH 3.4 to 5.0). Pepsin digestion in the stomach probably starts only in the last quarter of the first year, maybe somewhat stronger and earlier in naturally fed infants than in artificially nourished children.

The addition of lab to the milk is followed in the stomach by protein digestion. The lab ferment causes in the stomach a curdling of the milk (cow's milk at a pH of 6.0 to 6.4, breast milk at a pH of 5.0 to 5.5), and thus a separation of the whey from the casein fat curd. Only casein precipitated by lab contains calcium. Acid precipitated casein does not contain calcium. The rapid separation of solid and liquid components of the milk makes possible a rapid transport of the liquid part of the food through the pylorus. This prevents an excessive filling of the stomach in the course of a meal which is larger than the stomach capacity. The more important value of the lab coagulation seems to lie in the fact that milk fat is retained in the casein curd. Thus the digestion by the gastric lipase, the so-called esterase, which splits mostly the low fatty acids, can take place. The efficacy of gastric lipase is optimal at a pH of 4 to 5. The final result of the fat digestion in the stomach appears more far-reaching in the naturally fed child than the bottle fed child because breast milk itself already contains a lipase which is lacking in cow's milk. Milk lipase acts already in a pH of 8 to 6 where the digestive force of stomach lipase is still very low so that in case of breast feeding the work of the stomach lipase is assisted by the work of the milk lipase. Boiling of the milk delays lipolysis and thus also the emptying of the stomach since the fat contained in the stomach reflectively results in the closing of the pylorus.

The decisive influence upon the emptying time of the stomach, especially the delay of stomach emptying after cow's milk, originates most likely from the protein of cow's milk (casein and albumin) which by their quantity and quality inhibit the passage of the food into the duodenum.

The carbohydrate digestion is not continued in the stomach. Altogether, therefore, the digestive activity of the stomach is low in the infant. Of significance are only the digestion of fat and the separation of whey from protein whose digestion begins in the stomach. The undisturbed development of infants on whom in former times a gastro-enterostomy was performed as treatment for pyloric stenosis also points towards the fact that the importance of the stomach for the digestive process is relatively small.

The gastric digestion of the infant takes place in accordance with the following scheme:

1) Addition of lab ferment to the milk.
2) Coagulation by acid.
3) Partial protein splitting by cathepsin into albumin and peptone.
4) Fat splitting by lipase.
5) Beginning peptonization of protein.

The essential digestive action occurs beyond the pylorus, in the duodenum and the other sections of the small intestines. The effect of cathepsin in the digestion of protein continues. In addition all the ferments which are active in the adult also operate in the newborn and in the infant. The mucosa of the small intestines supplies already in the newborn secretin, enterokinase, lactase and maltase as effective ferments or as activators of ferment secretion. The pancreas furnishes amylase and maltase, a disaccharide splitting ferment, for the carbohydrate digestion. Trypsin and erepsin complete the protein digestion. Steapsin carries on the digestion of fats. The liver ejects already in the fetus bile into the intestines in which all the substances necessary for the digestion of fats have been found. The reaction in duodenum and small intestines, which increases from a pH of 5.0 to about 6.5 (in the lower ileum) corresponds to the range of reaction in which the most important protein and fat splitting ferments attain their optimal efficacy. In order to maintain this reaction, the artificially fed infant, because of the high protein content of his food which is acid binding, is forced to supply much larger quantities of intestinal secretion than the breast fed child. This increased secretory activity which also influences the intermediary metabolism through additional strain on water and salt metabolism is of greater disadvantage to the bottle fed child than to the breast fed infant. The final result of digestion in the small intestines is complete breakdown of protein; the final products show no essential difference between artificial and natural feeding. Furthermore, digestion of fat and splitting of lactose is completed in the small intestines. For this, the presence of iso-specific whey is more favorable than the presence of hetero-specific whey. The quantities of calcium and phosphate ions seem to be responsible for these differences. The inferior and more difficult splitting of lactose in case of artificial feeding is partly responsible for the greater tendency of bottle fed infants towards diarrheal disturbances since milk sugar which escapes digestion and is not completely absorbed favors the development of pathologic processes of fermentation (see later).

The digestion of nutritive substances through intestinal ferments which takes place in the large intestines is hardly sufficient to be of significance for the nutritive process. The main process taking place in the large intestines is an absorption of large quantities of water which up to then formed still part of the intestinal content. But even then the percentage of water in the stools of infants is still considerable, as seen in the following figures:

Normal stools contain	80% water
Hard stools contain	72% water
Diarrheal stools contain	88% water

The dry substances contained in the stools in addition to intestinal bacteria, intestinal cells and remnants of intestinal ferments are fat, ash and nitrogen in a relationship of 3:1:1/3 of the solid substances in the stool.

The fermentative digestion of food in the intestines, as described above, suffers no reduction even in cases of nutritional disturbances accompanied by diarrhea. Although the quantity of ferments might decrease they nevertheless remain present even in severest disturbances. This is the reason why all the frequent former attempts to find therapy with ferments for diarrheal diseases were found to remain ineffective since they tried to remedy a deficient function which did not exist. The only exceptions are those rare diseases which, due to organic changes, show a primary deficiency of a ferment (see cystic disease of the pancreas). The processes of digestion of food particles proceed in the intestines to such an extent that water soluble substances develop which can be absorbed by the cells of the intestinal wall and transported in the lymphatics or by the blood stream. Some of them are transformed in the intestinal wall and are made suitable for cell anabolism. Digestive fermentative forces of some tissues or body cells which might make possible a digestive process in a narrow sense beyond the intestinal tract, cannot be considered for practical purposes.

Bacteria and Digestive Process

The fermentative processes which condition the digestion of nutritive substances in accordance with physico-chemical laws are accompanied by processes of food decomposition through bacteria. To a small extent bacteria are always ingested with the food, to a larger part, however, they are permanently settled in certain limited territories of the intestinal tract. In the healthy infant the real digestive labour is performed exclusively by ferments of the stomach and the small intestines. In days of health the work of bacteria is limited to the lower sections of the intestines which are reached by only minor food remnants. However, the bacterial decomposition of food assumes decisive significance in case pathological processes disturb the normal equilibrium in the local distribution of fermentative and bacterial digestion. In that case the intestinal bacteria become sole rulers of food decomposition in the intestinal tract. This situation will be fully taken up in the description of the pathogenesis of acute diarrheal diseases. At this point we shall describe only the condition as found in the healthy infant.

While the oral cavity of the infant is germ free up to the time of birth, bacteria originating partly in the child's surroundings, partly in the food begin to infiltrate within a few hours. The broad connection of the oral cavity with the outside and with the cavities of nose and throat assures a constant change in the type of bacteria found in the oral cavity. This change, together with the constant flow of saliva and the mechanical cleansing of the oral cavity through sucking and swallowing motions make it impossible for a single type of bacteria to settle in the oral cavity. Only with a lowering of resistance of the organism the physiological cleansing becomes incomplete. As sign of this deficiency we might then find for instance the development of oidium albicans on the mucosa of the oral cavity. This development of thrush is possible in the newborn and in the young infant already on the basis of minor damages favored by the relatively low salivary secretion. In the older infant, however, the appearance of thrush is a symptom of serious damage to the immunity of the mucous membranes. In general the behaviour of oral mucosa depicts the processes (in the pathogenesis of acute diarrheal disease) of the mucosa in the stomach and small intestines.

The amount and type of bacteria in the stomach are essentially determined by the bacterial content of the food. The number of germs in the stomach is small in the healthy child, perhaps because the hydrochloric acid in the stomach and even the not excessively high hydrogen ion concentrations of the gastric juices of the infant have a bacteriocidal or bacteriostatic effect. In the case of infants fed with cow's milk the loss of acid caused by the stronger binding power of the larger protein quantities of the casein curd (as compared to the lower protein content of breast milk) is of significance for the development of a number of intestinal disturbances in the artificially fed infant since loss of acid is accompanied by a decrease of the germ killing force of the gastric contents. If gastric functions take their normal course the normal reactions of the gastric juices are sufficient to prevent an unchecked growth of the germs. The types of bacteria present in the stomach of the healthy infant are: streptococci, enterococci (streptococcus faecalis), yeasts and not infrequently also bacillus coli (Escherichia coli). Whether small quantities of bacilli coli in the stomach can be of importance for the development of diarrheal diseases is doubtful. Some authors assume that the coli bacilli which are found in the stomach in cases of nutritional disturbances had risen from the duodenum into the stomach or that they are forms with special characteristics which can exist in the stomach. In case of very unclean food, rich in bacteria, it is, however, probable that the settling of coli in the stomach can become the point of origin of a bacterial invasion of the small intestines. With the appearance of an acute diarrheal disease the normal relative dearth of germs in the stomach is replaced by a colonization of germs of the coli group. In the healthy infant the flow of gastric juices and the regular emptying of the stomach, similar to the activities of the oral cavity, take care that bacterial decomposition of the food in the stomach cannot take place to any significant extent. The prerequisite for this is a stagnation of the contents of the stomach over a longer period of time.

The small intestines, even more than the stomach, are bacteria free in the healthy child. This applies at least to the duodenum and jejunum. From the large intestines, which are thickly settled with bacteria, a few germs always travel into the lowest sections of the ileum so that we constantly find there bacteria in larger quantities. The upper sections of the small intestines show only isolated enterococci, bacteria of the lactis-aerogenes group (lactobacillus acidophilus) while only in the lower sections of the ileum the bacillus coli begins to play a larger role. The large intestines beginning with Bauhin's valve down to the rectum are inhabited by numerous germs of different types. Apart from fermentation causing bacteria (enterococci, bact. aerogenes etc.) which are scarce in the small intestines and abundant in the large intestines there exists a bacterial flora which in its composition is determined by germs in the food which occasionally reach the lowest sections of the intestines.

The meconium, which at the time of birth is sterile, is settled by anaerobic sporogenes bacteria which are rapidly replaced by the real intestinal bacteria, probably because of the change in intestinal reaction. If the food consists of breast milk an almost pure culture of gram-positive lactobacillus bifidus develops in the large intestines which constitutes 90% of the entire stool. The abundance of these bacilli places all other germs into the background even though they never disappear completely. It is therefore possible to find, although only in very small number, bacillus acidophilus, enterococcus, bacillus coli, proteus vulgaris and staphylococcus; all these are germs which under special circumstances can become pathogenic. For the development of lactobacillus bifidus a number of factors must coincide which are encountered only in breast milk. Perhaps a substance, as yet unknown, in the breast milk whey is here of decisive significance. In case of artificial feeding (for instance with partially predigested cow's milk) special and complicated conditions must be fulfilled to produce a bifidus flora. It has not been proven whether the development of a bifidus flora in case of artificial feeding can be regarded as criterion that artificial food thus approximates natural food in value.

While the gram-positive flora of a single bacteria in an almost pure culture is characteristic for the breast fed child the flora in the artificially fed child is gram-negative, consisting essentially of bacillus coli and bacterium lactis aerogenes (lactobacillus acidophilus). In addition, two thirds of the infants carry spore-producing bacilli, paracoli, anaerobic bacteria, bacterium proteus, bacterium alkaligenes, bacterium pyocyaneum (Pseudomonas pyocyanea), clostridium etc. The picture of bacterial flora in the large intestines is more varied in the artificially fed child than in the breast fed child. Only about one third of the children fed artificially with cow's milk show bacillus coli and bacterium aerogenes exclusively in the stools. Bacteria of the paracoli group which are perhaps of significance in the pathogenesis of acute diarrheal diseases, are found in 22% of all healthy infants. The constant presence of large numbers of coli bacilli in the large intestines, next to the digesting sections of the small intestines, is one of the reasons why the bottle fed infant shows more inclination toward acute diarrheal diseases which are always accompanied by an ascent of bacteria in the small intestines, than the breast fed child whose entire small intestines are, in days of health, almost free of coli and paracoli bacteria. In the last analysis, however, it is the regular emptying, effected by proper functioning of motor and cleansing mechanisms, the peristalsis, which is of decisive significance for the absence of germs in the small intestines as well as in other organs of the body (bladder, bronchi, etc.). A disturbance in this mechanism (chymus-stagnation) is the first step leading to the settling of germs in sections of the intestinal tract which physiologically contain few germs.

The action of special substances which check the development of bacteria and contribute to the permanence of the absence of germs in the small intestines (so-called bacteriostanine, bacteriolysine) is as yet little explored. However, the existence of such vital substances is probable.

Formerly the germs in the intestinal tract were divided in accordance with their functional achievements into those causing fermentation and those causing putrefaction, with the point of action of the fermenting bacteria being essentially the carbohydrates, with that of the putrefying bacteria essentially the protein bodies. The fermenting bacteria included the enterococci, the lactic acid forming bacteria (for instance bacillus bifidus), the groups of coli bacilli and lactis-aerogenes and finally bacteria which are able to form butyric acid from carbohydrates. As the main putrefying bacteria were considered the anaerobic and facultatively anaerobic bacteria; but in this group, too, appeared the bacillus coli. This classification alone shows that a sharp division of bacteria into those causing fermentation and those causing putrefaction does not correspond to the facts. The important bacillus coli which shows no preference with regard to its medium has in any case the ability to accomplish both types of food decomposition.

In the germ-free small intestines of the healthy child the bacteria do not participate in food decomposition. Germs assume significance only in the large intestines. Here the small quantities of unabsorbed chyme are attacked and decomposed by the types of bacteria in accordance with their abilities. The food remnants are joined by not inconsiderable amounts of protein-rich intestinal juices which were not re-absorbed and thus become subject to bacterial decomposition. The protein content of these food remnants, especially upon encountering calcium and magnesium, favors the development of proteolytic bacteria. Carbohydrates which reach the lowest sections of the intestines unsplit or unabsorbed promote here the fermenting processes. The various types of sugar react somewhat differently: lactose and malt extract are more subject to fermentation than cane sugar and polysaccharide mixtures; wheat flour less than darker types of flour (rye, oats). Fats (fatty acids) seem to promote the development of fermentation more indirectly, not by reinforcing the fermenting processes themselves but by supporting the development of carbohydrate fermentation in the struggle with putrefaction. In general it can be assumed that processes of fermentation and putrefaction always take place simultaneously in the large intestines and partly also in the lower ileum. "Normally the intestinal content is subject to lawful bacterial growth which changes according to plan. The location and changes of the bacterial flora are dictated not only by the accidental presence of bacteria but still more important by the type of the medium in which they grow" (*von Noorden*).

The extent of the processes of decomposition and of fermentation in the intestinal tract influences the reaction of the stools: acid feces can be considered fermentation stools and alkaline feces putrefaction stools. On testing with litmus paper the stools of the healthy breast fed infant show an acid reaction, those of the healthy bottle fed child an alkaline reaction. Acceleration of peristalsis especially in the small intestines, as seen in diarrheal diseases, changes the stool reaction of the artificially fed child which now becomes acid. Occasionally, however, in case of excessive secretion of alkaline intestinal juices which in the course of serious diarrheal diseases reach the large intestines, alkaline reacting stools may result.

The biological significance of the bacterial flora in the intestinal tract for the basic digestive processes of the healthy organism is not yet determined. It is probable that even in the absence of bacteria from the lowest sections of the intestines the nutritional processes would take a normal course. The results of the new drugs (sulfonamide preparations and antibiotics) which lead practically to an absence of germs even in the large intestines, have shown that "normal" intestinal bacteria constitute a protection for the organism against the invasion and growth of other germs. The observation of the appearance of staphylococci in the large intestines and the development of general staphylococcic diseases after the removal of normal intestinal inhabitants through occasionally successful "amicrobiosis" of the large intestines, proves the biological value of physiological intestinal flora in certain sections of the intestines. The bacteria of the large intestines are also of significance for the production of vitamin K. The formation of intestinal gases and fermentation acids is of significance for the processes of food absorption, stool formation, peristalsis and stool elimination.

The organism of the infant is engaged in a constant struggle to limit the settling of bacteria to territories where no vital phases of fermentative digestive processes take place. The young infant is not well adapted to this struggle. Some of the harmless diarrheas of the first period of life might be an expression of the difficulties which the infant faces in order to adjust to the presence of the necessary bacteria.

"In the intestinal tract, especially below the lowest ileum, a vegetative microbic life exists, the harmlessness of which is based not on the absence of toxic bacteria but on the existence of an orderly equilibrium among the different species of the flora. . . Foreign intruders can be the first cause for disturbing this equilibrium. When the intruders themselves assume leadership, when they displace other germs through the force of their virulence (as e.g., in cholera) or when they endanger the organism by their specific poisons, even without excessive increase in number, we speak of infectious diseases of the intestinal tract" (*von Noorden*). The problems of pathology of the bacteria in the intestinal tract will be discussed in full in the chapter on the pathogenesis of acute diarrheal diseases and infectious intestinal diseases.

ABSORPTION OF FOOD AND INTERMEDIARY PROCESSES

Metabolic tests give information regarding the processes taking place in the absorption of food in the intestinal tract, the consumption in the organism for energy and anabolic metabolism and the elimination of food remnants. The results of these tests show only the quantity of absorption, the utilization of food and the quantities of waste. The tests give, however, little information regarding the quality of processes from the moment the food enters the circulation until its anabolism and catabolism in the cells, and the elimination of the remnants of the digestive process in the excretions. Facts regarding the intermediary metabolism in the organism are, therefore, little known. The largest quantities of oxidizable substances (protein, carbohydrate and fats) are utilized to the greatest extent for vital processes. A smaller part serves the processes of growth and the replacement of dying cells.

Absorption of nutritive substances does not take place in the stomach but in the entire small intestine. Here, protein breakdown products (polypeptides and amino-acids), carbohydrates split into monosaccharides and fats (mostly neutral fats), and also all salts and vitamins, together with large quantities of water are absorbed. The absorbed particles reach the main circulation and from there the tissues by way of the blood and lymph vessels, with a detour through the liver or (like the majority of fats which are again rebuilt in the intestinal wall) directly by way of the lymph passages. The only process taking place in the large intestines is essentially an absorption of water leading to a thickening of the hitherto liquid chyme until it reaches the consistency of stools. Apart from the considerable absorptive process there are in the small intestines—to a lesser extent also in the large intestines—strong currents which carry liquid, salts, protein, etc. from the circulation back into the lumen of the intestine. This secernent ability of the small intestines, consisting mainly in the secretion of intestinal juices, is considerable. The quantities of liquid eliminated into the small intestines from the adjacent glands of the intestinal tract (liver, pancreas) and from the intestinal glands themselves exceed by far the amounts of liquid taken in with the food. The amount of saliva, gastric and intestinal juices, bile and pancreatic juice which flows into the small intestines in the course of a day can only be roughly estimated. By the time a certain quantity of milk intake reaches the pylorus the amount of liquid is probably increased by half (with a milk intake of 200g, the increase is about 80 to 100g). In the small intestines another 150g are added so that with every meal of 200g of milk another 250g of liquid are supplied which partly consist already of the rapidly absorbed water of the meal. If the child takes in five meals a day the water metabolism (absorption and secretion) amounts to about $1^1/_2$ liter a day. In addition there is the constant secretion of saliva, gastric and pancreatic juices, etc. which continues even between meals and which is estimated at about one-half to one liter a day. Thus, an infant taking in one liter of food a day (milk, cereal etc.) would metabolize altogether two to three liters of water. Since about one-third of ingested liquid is eliminated as insensible perspiration and the remainder, except for small amounts which are used for growth is lost in form of urine or stool, it follows that every part of ingested water must complete the course between intestines—circulation—intestines at least five times a day. This adjustment of water metabolism (great performance with small means) makes it understandable that even small losses of water with increased perspiration through lungs or skin, as seen in all diarrheal diseases, must be noticeable in the intermediary metabolism. Insufficient thickening of food remnants in the large intestines, as takes place in diarrhea, is much less disturbing since these losses of water in the stools are to a large extent balanced by decrease in urine quantities and elimination of concentrated urine increased to the point of anuria, if necessary.

The absorption of mineral substances (Na, K, Cl, Mg, Fe, Phosphate) takes place in the small intestines. The salts are ionized in the body. Part of the ingested salts leave the organism, when no longer needed, by way of the kidneys (Na, K, Cl, Phosphate), while other salts (compounds of Ca, Mg, Fe), after being absorbed by the small intestines and having passed through the circulation, are again eliminated in the large intestines. The salts which are eliminated in the course of a day through urine and stools are, of course, in no way those which were ingested during that same day. It can be assumed that the salts in urine and stools are end products of regressive processes (e.g., in the bones), which are not in direct connection to processes of intake.

The daily retentions for the most essential mineral substances (the so-called utilization value) amount in the newborn to the following values:

P_2O_5	170–250 mg
CaO	180–290 mg
Cl	95 mg

Long term investigations by special methods have yielded the following values for the total ash balance in the infant with different types of nutrition:

	Daily intake (gram)	*Retention*	
		Absolute (gram)	*% of intake*
Breast feeding	1.07–1.5	0.37–0.72	32–48%
Bottle feeding	2.46–5.8	0.83–3.8	24–65%
Mixed feeding	2.33–3.9	1.12–1.3	33–48%

The artificially fed child retains thus more mineral substances (especially Na and K) than the breast fed child. Whether this hyper-mineralization of the bottle fed child is of biological significance is as yet not determined. It must be considered, however, that about 10% of sodium and chlorine are eliminated through the skin (perspiration). It is very probable that this hyper-mineralization of the bottle fed child is occasionally balanced by larger losses.

The balances of the different mineral substances, as seen in metabolic tests, show considerable variations depending on the age of the child and the food composition. The results of these investigations are more of theoretical than practical interest. Their description can therefore be omitted at this place.

Protein bodies are to a large extent broken down into amino-acids and absorbed as such or to a smaller extent as polypeptides. Passage of the entire protein molecule through the intestinal wall does not take place in the infant, except during the newborn period. It seems that in the first days of life ferments, hormones, antitoxins and similar biologically important substances and (at least in the young animal) a number of protein substances, perhaps also micro-organisms (tubercle bacilli) penetrate the then still permeable intestinal wall.

Protein, which is absorbed in the form of its basic elements, is again built up into complex compounds, partly already in the intestinal wall, partly in the liver. Hardly any facts of practical importance are known about the further passage of the proteins to the cells. The greatest part of ingested protein is absorbed and used for anabolism. In breast fed infants absorption should be almost 100%, in bottle fed infants somewhat less. The amounts of nitrogen which appear in the stools should not be regarded as remnants of ingested food protein. They originate essentially from the protein of the intestinal juices and the intestinal bacteria.

The utilization value of protein, calculated on the basis of the amount of nitrogen utilized by the body for the building of tissues, varies between 78.3% in the newborn and 23.2% in the five months old breast fed child or, in case of artificial feeding, between 37.6% in the second month and 18.8% in the sixth month. The slowing down in growth explains this gradual decrease in quantities of anabolized protein.

The fat, after its digestion in the intestines is, similar to the protein, absorbed and again built up in the intestinal wall through cell activity. The fat droplets which are emulsified in the blood make the blood serum opaque on their way through the organism (Lipemia), although the degree of opaqueness does not correspond to the quantities of circulating fat. The amounts of absorbed fat are subject to marked variations depending upon the other food components and upon processes in the gastrointestinal tract. High fat content of the food causes a relative decrease in absorption. Some types of diarrhea have a similar action. This disturbance in the fat absorption in the presence of a well balanced food intake is particularly pronounced in the case of celiac syndrome. In this disease 40 to 50% of the stools (compared to normal figures of 10 to 30%) consist of fat. The unabsorbed fat appears in the stools as neutral fat, fat soaps and fatty acids.

Carbohydrates are ingested as monosaccharides. With a normal functioning of the gastro-intestinal tract absorption approximates 100%, provided the infant's relatively high limit of tolerance for sugar is not exceeded. Other food components inhibit the absorption of sugar: water and breast milk whey the least, protein and fat somewhat more, and cow's milk whey the most, where the protein content, next to mineral substances (phosphate) causes a delay in sugar absorption. On the other hand, the increase in carbohydrate intake in a certain diet effects a better protein balance, namely the nitrogen content in the urine decreases considerably and does not increase correspondingly in the stools.

Liquids, particularly water, are according to present-day concepts stored in three systems which are separated from each other by a surface which acts like a semi-permeable membrane: that is, intracellular fluid, interstitial or tissue fluid and the fluid in the blood stream which is contained in the plasma. Water and salt ions penetrate through these dividing layers. The passage of water from one system into an adjoining one is furthered through the migration of salts or rather their ions, of which Na, K and Cl are the most important. The organism consists of about 70% of its weight in water, of which in the infant about 45% are stored within the cells, 20% in interstitial tissues and 5% in the blood vessels as plasma. In the healthy individual all these systems are in a state of osmotic equilibrium. The constancy of salt (ion) contents provides for the constancy of osmotic pressure, although not the same salts are stored in the different systems. The main difference in the distribution of salts lies in the arrangement of K and Na. The major part of potassium is found in the cells. In addition the intracellular fluid, in contrast to the extracellular fluid, contains calcium bicarbonate, glycogen, etc. The largest quantity of sodium is stored in the interstitial fluid and in the plasma, where it regulates osmotic pressure.

The fluid distributed in the different systems according to physical laws is of varying biologic value for the organism. Water binding in the extracellular space, mainly in interstitial tissue, is probably relatively simple. It is a reservoir placed as a buffer and protection between the vital intracellular fluid and the fluid in the blood stream which provides for the water metabolism. But even in the cellular fluid and in the plasma part of the water is used in the building of the large protein molecules which are found there. In all parts of the body water is present in three different combinations, as construction water, adhesion water and free water. The free water is retained in the meshes of the micellae from which the colloids of the protoplasm are built (see p. 22). The adhesion water is already bound stronger in the colloids even if it does not yet determine decisively the structure of the cells. The construction water is an essential component of living substance which, if removed from the tissue, causes a serious and often irreparable damage to the cells. The majority of disturbances in water metabolism which will be mentioned repeatedly in the following pages, concern the free water and the salts contained therein. Only in the most serious forms of acute nutritional disturbances are adhesion water and construction water also affected. The diverse and complicated demands on the water metabolism which occur in daily life are also essentially regulated by free water. This applies to catabolism and anabolism of nutritional substances and elimination of waste materials through kidney, intestinal tract, lungs and skin.

In days of health and disease the organism strives to maintain a constant osmotic pressure in the blood (plasma). The large quantities of colloids and lipoids in the plasma prevent in the healthy individual the efflux of liquids through the capillary walls. If some damage causes a decrease in pressure in the extracellular space which cannot be balanced immediately by the kidneys, the organism attempts to bring about an equalization of pressure by utilizing tissue fluid and finally intracellular fluid. Fluid from the intercellular and even the intracellular space travels through the semi-permeable membranes. The result is a loss of salts, primarily of Na and Cl, or finally of K from the cells which are lost for the body to the same extent as the kidney eliminates them in the urine. Similar to water losses from the plasma, salt losses through the intestines (diarrhea) or through the kidneys can lead to displacement and loss of sodium from the tissues or potassium from the cells. Here, too, disturbances in osmotic pressure are as far as possible evened out by the organism, e.g., by the entrance of Na in place of K into the cells.

Little is known about the intermediary metabolism of food between the intestines and the organs of elimination. It is, however, a definite and practically important fact that the process of intermediary metabolism of water and vital salts is essentially determined by the reaction of the juices and the tissues. Disturbances in the acid-base balance have been proven in a large number of pathological features and in many well known disease entities.

Deviations in the acid-base balance from the norm toward the acid side are referred to as acidosis and toward the alkaline side as alkalosis. Acidosis defines a state which shows primarily an accumulation of acid ions (anions) or secondarily a loss of alkaline ions (cations). On the other hand, an alkalosis develops from an accumulation of alkaline ions or loss of acid ions.

According to the mechanisms which cause the state of acidosis

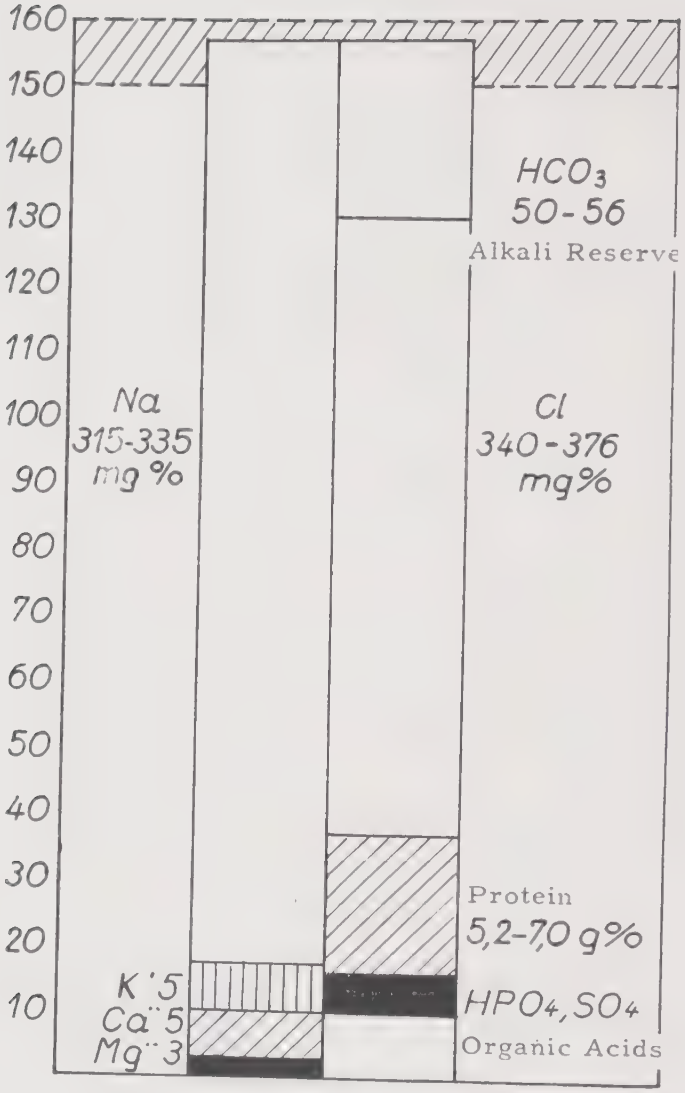


Fig. 9. Acid base balance (J. L. Gamble) in blood plasma in healthy infants. In milliequivalents per liter we find:

Base:		Acid:	
Na	142	HCO_3	27
K	5	Cl	103
Ca	5	HPO_4	2
Mg	3	SO_4	1
	155	Org. Acids	6
		Protein	16
			155

or alkalosis, respectively, the following conditions are differentiated:

Alkali excess	= Metabolic	Alkalosis
Alkali deficit		Acidosis
CO_2 excess	= Respiratory	Acidosis
CO_2 deficit		Alkalosis

One refers to compensated or non-compensated acidosis or alkalosis, depending on whether the pH of the blood is normal or abnormal. The organism attempts to maintain the constancy of reactions by the buffer system which it has at its disposal.

The measuring of acidosis takes place by determining the CO_2 combining power of the blood and, corresponding to the definition of acidosis, a decrease of bicarbonate in the blood to below the norm is found. Normal blood has, at a CO_2 tension of 40 mm in the alveolar air, a CO_2 combining power of 40 to 60 volume per cent. Any drop below 40 volume per cent is to be regarded as sign of acidosis. Acidosis and alkalosis, as far as they are significant for questions of nutritional disturbances in the infant, develop in the following manners: acidosis through primary losses of alkali, through increase of intermediary developing acids in case of prolonged hunger periods or through losses of alkali in repeated and prolonged vomiting, accompanied by losses of duodenal juice, and above all in strong diarrheas. A CO_2 accumulation in states of asphyxia due to heart failure also leads to acidosis. Alkalosis is brought about through accumulation of alkaline ions, as a result of vomiting in pyloric obstructions (pylorospasms) through loss of acid gastric juices, in intestinal obstruction where large quantities of water and chlorides are lost or as a result of large CO_2 losses in case of hyperpnea, for instance in high fever or encephalitic processes with intact circulation which exist not infrequently in nutritional disturbances (e.g., salmonella infection). It is important, however, to avoid the erroneous assumption that there is a strong contrast between alkalosis and acidosis, as in the sense of a total change of reaction. It is only a question of change in the reaction of the body liquids, measured by the contents of H ions in the blood, the urine, etc.

The concentration of hydrogen ions in the plasma is dependent on the relationship carbonic acid: bicarbonate. A quantitative proportion of H_2CO_3:$BHCO_3$ of 1:20 guarantees a normal pH of 7.4. The organism attempts to maintain this physiologic reaction of the blood as constant as possible.

Two regulatory systems are at the disposal of the organism for maintaining a certain reaction in the blood and in the tissue fluids: through raising or lowering carbon dioxide elimination through the lungs or through elimination of electrolytes (Na′, Cl′ phosphates) in the urine.

If the body is endangered by displacement of the normal reaction of juices toward the acid or the alkaline side, either through invasion or through increased formation of acids or alkalis, the organism attempts to compensate this danger by changes in the breathing process or by changes in kidney secretion. By altering the type of respiration (deep, rapid breathing) an excess of carbon dioxide can be removed from the body. A change in kidney secretion, mainly through increased or decreased elimination of primary or secondary phosphates which is connected with the elimination of sodium or potassium also prevents a threatening displacement of reaction toward the alkaline or the acid side. These compensating mechanisms operate until the normal reaction of the blood has been re-established. In cases of increase of carbon dioxide in the blood the normal relationship between CO_2 and $NaHCO_3$ is again established through rapid and deep breathing. Through superficial or slow breathing CO_2 is retained in the blood and thus an excess of $Na\,HCO_3$ is again compensated. Since normally the reaction of the urine is essentially determined by the quantitative proportion of primary to secondary phosphates, a compensation of deviations of balance in the reaction of tissue juices can be effected in a similar manner also from that side. If, for instance, increased amounts of acids appear in the organism, part of the secondary phosphate in the blood turns intermediarily into acid, primary phosphate. This excessive formation of primary phosphate stimulates the kidneys to elimination, and the quantities withdrawn from the blood into the urine are such as to re-establish intermediarily the normal relationship of primary to secondary phosphate (in a pH of 7.4 about 1:4). The efforts of the organism to maintain the reaction of tissue fluids as constant as possible by altering the quantities of acid or alkaline ions in the products of elimination, show that the reaction of metabolic processes can be determined by judging the acid and alkaline products in the eliminations (carbon dioxide, phosphate) or by establishing the bicarbonate in the serum.

A comparison of the most important data of the acid base balance, which in some respects are opposite in the breast fed and the bottle fed child, can be summarized in the following table:

		Breast Fed	*Bottle Fed*	*Comparison* *Breast Fed to Bottle Fed*	*Comparison* *Bottle Fed to Breast Fed*
Blood	pH	7.30	7.33	same	same
	Plasma CO_2 (Vol,%)	53.2	44.3	higher	lower
	Plasma carbonate number	1.3–1.78 (1.59)	1.33–1.48 (1.39)	higher	lower
Urine	pH	abt.5.5	abt.7.0	decreased	increased
	Titrable Acidity	4.0–13.9 (9.3)	10.5–72.4 (50.0)	decreased	increased
	Ammonium (n/10 cc)	22.4–69.1 (45.2)	39.9–142.7 (80.7)	decreased	increased
	Organic acids (n/10 cc)	10.8	13.8	decreased	increased

This table shows that the metabolism of the healthy breast fed child, compared to that of the healthy bottle fed child, has a tendency towards alkalosis. Infants fed with cow's milk thus show a predisposition toward acidosis.

The normal composition of the blood serum for the first year of life, beyond the newborn period, can be described with the following data:

Total Protein	5.33–6.94	g%
Fibrogen	0.26–0.4	
Albumin	3.79–4.7	
Globulin	1.31–2.0	
Non Protein Nitrogen	25–40	mg%
Urea Nitrogen	19–40	mg%
Amino-acid Nitrogen	4.8–7.8	mg%
Uric acid	1.0–4.0	mg%
Creatinin	0.9–2.0	mg%
Blood sugar	80–120	mg%
Chloride (Blood)	450–550	mg%
Sodium Chloride	570–620	mg%

Continued

Cholesterol	87–180	mg%	(150 mg%)
Sodium	2.9–4.07	mg%	
Potassium	0.19–0.38	mg%	
Calcium	9–11.5	mg%	(10.5)
Phosphorus (anorganic) in Plasma	4.8–6.8	mg%	(5.4)
Phosphatase (acid)	1–3	U	
Phosphatase (alkaline)	5–10	U	
Acetone, Acetoacetic acid	Traces		

EXCRETIONS

The waste materials of the metabolic processes are eliminated through urine, stools and insensible perspiration through skin and lungs. In addition to water urine contains mainly the waste products of protein metabolism, alkalis and phosphates; the stools contain the amounts of fat and carbohydrates which escaped digestion, as well as calcium, magnesium and iron; the largest amount of water leaves the organism through insensible perspiration and through the kidneys.

URINE

With the exception of the first days of life when the urine is rich in urates and occasionally of a reddish color, the urine of the infant is pale yellow, clear, and of very low specific gravity. In the artificially fed child the urine reaction is acid due to the large quantity of acid waste products developed in the breakdown of the relatively large quantities of protein. In the breast fed child the reaction of the urine is alkaline, almost neutral. The waste products of protein metabolism appearing in the urine are: urea (60 to 80% of total N of the urine), ammonium and non-protein nitrogen which also includes some amount of amino-acids. The amount of ammonium in the urine nitrogen (ammonium coefficient) shows wide variations. Its size has for a long time been an indication for the direction of the entire metabolic process. In case of stronger acidosis the ammonium elimination in the urine increases. Further present in the urine are part of the sodium and potassium, to a large extent bound to chlorine.

As a peculiarity of newborn metabolism relatively large quantities of urates and nucleo-albumins are found in the urine in the first days of life due to the diet of colostrum which is rich in protein and cells. These substances cause the so-called uric acid infarct of the kidney which develops as a result of low water intake during the newborn period and which upon elimination causes the reddish color of the urine in many newborns. Lack of fluid intake can also result in the so-called albuminuria of the newborn.

MECONIUM

The first stool, which is emptied already during or immediately following birth, is the meconium. Meconium is almost odorless, of dark green to greyish-black color caused by its content of biliverdin and bilirubin which are not, as in normal stools, decomposed through bacterial action into hydrobilirubin. Meconium is of thick, viscid consistency and has a shiny appearance. It contains no bacteria. Meconium consists mainly of intestinal cells, skin cells, lanugo hair and fat from the vernix caseosa which was swallowed by the fetus during pregnancy. Remnants of the epithelium which were left over after the canalization of the intestinal tract during the third fetal month form an essential part of the meconium. The amount of salts contained in the meconium is relatively low. The amount of water is relatively large compared to the normal stools of later life. Ten to 15% of dry substance, that is, $^1/_3$ to $^1/_5$ of the total quantity of meconium, consist of fatty acids and cholesterol which originate in the vernix caseosa and the skin fat of the fetus. Fat content amounts to about 10% of the total quantity. Nitrogen containing substances amount to about 5 to 6% of dry substances. The origin of the relatively small quantities of polysaccharides, among them many non fermenting mucopolysaccharides, in the meconium is as yet unexplained.

STOOL

The stools of the infant always contain, in addition to water, protein, calcium and magnesium salts, iron, fats, waste products of the metabolism and carbohydrates in greatly variable quantities. The color of the stools in children fed mainly with milk originates almost only from the admixture of bilirubin. Urobilin is almost completely absent in the stools for a long period after birth. The odor of the stool in breast fed infants is determined by fatty acids and oxy-fatty acids. In the bottle fed child the odor of waste products of protein (decomposition) is added. The protein content of the stools (about 4 to 5% N of the dry substance) consists to the smallest extent of food remnants, to a larger extent of nitrogen containing intestinal secretions and bacteria.

> Formerly the greyish-white or white "milk particles" which appear in the stool in varying forms were regarded as unabsorbed protein; because of their supposed origin they were described as "casein particles." After long controversies it can be considered certain that the "milk particles" are only to a small extent undigested remnants of milk protein. The small, white, easily crushed particles which appear in large quantities in diarrheal, mucous stools are essentially fat soaps. Their nitrogen content is low. The larger, tougher forms which are often elongated, of grey color and which appear very infrequently in the stools consist, on the other hand, mainly of casein and para-casein with only little admixture of fat soaps and fatty acids. The large, tough particles are found regularly in the stool only in children fed with raw milk.

The fats of the stools consist of neutral fats and free high fatty acids, of water soluble alkali soaps and water insoluble alkaline earth soaps. The more consistent the stool the higher its content of calcium and magnesium salts of fatty acids. With increased fat content of the food and lowered absorption the fat content of the stool increases. With artificial feeding mixtures which are rich in fat or with poor absorption of fats (e.g., in celiac disease) it is occasionally possible to rediscover in the shiny stools 60 to

COMPOSITION OF FECES IN CHILDREN FED WITH MILK

Substance		*Breast Milk (%)*	*Cow's Milk (%)*
Water		81.22	78.82
Solid substances		18.78	21.18
Ash	per 100 cc milk	11.0	15.0
Water insoluble salts		9.5	13.2
Water soluble salts		1.5	1.8
Total Nitrogen	per about 100 parts Dry substance	4.5	5.9
Protein substance		20.6	30.6
Fat and Fatty acid soaps (Cholesterol, Cholic acid etc.)		40.0	40.0

70% of the ingested fat. Carbohydrates (lactose) are almost completely absent in the stools of healthy infants. Polysaccharides are not found in the stool in larger quantities in artificially fed children with normal speed of the peristalsis and therefore complete digestion in the intestinal tract. If the peristalsis is accelerated, however, undigested sugar and starch are abundant in the feces. Undigested flour can easily be traced through the iodine test.

INSENSIBLE PERSPIRATION AND WATER METABOLISM

The elimination of water which is left over from the metabolic processes takes place in the infant through three channels: stools, urine and insensible perspiration through lungs and skin. The quantities of water eliminated in the normal stool are small: they amount to only about 3 to 5% of the intake. In case of diarrhea the amounts of water eliminated in the stool can increase to 200 to 500g per day. These excessive water losses in the stools are essentially compensated by saving of water in the urine. The remainder, about 35 to 40% of the total water intake, leaves the organism with the exhaled air and through the skin. Under normal conditions the amount of water eliminated per square meter of body surface is about 15 to 16g in the newborn, 16 to 19g in the older infant. The extent of insensible perspiration in the infant is far more dependent on the quantity of fluid intake than that of the adult. However, the relationship between urine and insensible perspiration shows great variations in the young child. A number of external factors can change the method by which the water leaves the body. Crying, restlessness, fever, climatic factors, concentration and composition of food (protein content) increase the elimination through skin or lungs. But above all water retention and water elimination, as previously mentioned, show rhythmic changes within the twenty four hours of the day. In the course of a day the infant gains 150–200g in weight; of this increase 120 to 170g are lost again during the night.

NUTRITIONAL REQUIREMENTS

In order to determine the nutritional needs of an infant it is sufficient for practical purposes to indicate the quantities of milk, sugar, etc. which an infant of a certain age and weight requires. An approximate indication for the artificially fed child is the quantity of milk taken by the well developing breast fed child, provided that the calories of the milk mixture and the nutritive substances contained therein correspond in quantity and quality approximately to those of breast milk. The quantities of milk consumed by the breast fed child amount to about $^{1}/_{5}$ of the body weight during the first weeks of life, later, until about the sixth month of life, $^{1}/_{6}$ to $^{1}/_{7}$ and in the second half of the first year about $^{1}/_{8}$ of the body weight; that is, the quantities which the infant, at his own discretion, sucks from the breast amount to about 500 to 600g in the first three months of life, 700 to 800g in the second three months, and about 1000g in the second half of the first year. But even in the breast fed child deviations from the "normal figures" are not infrequent. There are a number of children who thrive excellently on much less milk intake while on the other hand many children taking larger quantities do not appear overfed. For children fed with cow's milk it is much more difficult to establish similar figures with any degree of exactness because the nutritive substances contained in cow's milk vary greatly due to care, feeding and breeding of the cow. The nutritive value of milk varies considerably because of the customary dilutions and additions in the preparation of infant food. The approximate amounts of food intake consisting of $^{1}/_{10}$ of the body weight in milk plus $^{1}/_{100}$ in sugar should give satisfactory results. In following these indications the total quantity of food intake (milk plus addition of diluting fluid) would follow the intake of the breast fed child. This also covers the large fluid requirements of the infant.

Only by calculating the nutritional requirements from the caloric values of the food, as shown by *Rubner*, *Heubner* and *Camerer*, is it possible to give more exact indications for the nutritional requirements of the infant. But here, too, it must be remembered that there is no absolutely valid figure for the nutritional requirements of the infant. External and internal factors can affect the nutritional requirements considerably. We might mention here the surprisingly high increases caused for instance by influences of the glands of inner secretion, restlessness, or lively crying. Prolonged crying of an infant can raise the nutritional requirements by 100% or more.

The quantities of calories required per kilogram of bodyweight were referred to by *Heubner* as energy quotient. The energy quotient in the young breast fed child lies around 100, in the older breast fed child around 90, in the second half of the first year around 80 and toward the end of the first year of life around 70 calories. The energy quotient of the artificially fed child was assumed already by *Heubner* to be higher than in the breast fed child, at about 120 calories per kg bodyweight. Assuming an approximately even composition of cow's milk the knowledge of the energy quotient makes it possible to determine the requirements of intake of a certain feeding mixture from the known body weight of the infant.

1 liter whole milk	about 600 calories
1 liter $^{1}/_{2}$ milk plus 5% sugar	about 500 calories
1 liter $^{2}/_{3}$ milk plus 5% sugar	about 600 calories
1 liter buttermilk, 1% flour & 6% sugar	about 600 calories
1 liter protein milk plus 5% sugar	about 620 calories

The determination of the nutritional requirements of a child from the caloric value of the food and the body weight of the child is subject to a number of errors which can cause disturbances in the practice of nutrition if the physician adheres too strictly to the average values of caloric requirements. The figures for the nutritional requirements are averages which must be utilized only for mass feedings or for calculating the nutritional requirements of the entire population. For the individual child deviations from the "standard figures" to either side are frequent. Already during infancy, certainly after the third week of life, we find individuals with higher or lower caloric requirements, similar as in the adult. In addition there is another limitation as to the exactness of this method: the calculation of nutritional requirements on the basis of body weight is basically inexact.

The exact procedure is a calculation of caloric requirements not on the basis of body weight but of body surface. The reason why the body surface is the physiologically correct basis for calculating the nutritional requirements is because the loss of heat takes place essentially according to the extent of body surface. It is not deter-

> mined decisively by the body weight. The amount of heat which is lost on the body surface through radiation and evaporation of water must be replaced by food intake. The reason for discontinuing the calculation of nutritional requirements on the basis of body surface was essentially a concession to the practice of pediatrics since the determination of body surface is complicated, that of body weight extremely simple.
>
> The following observation shows that body weight does not go parallel with loss of heat: in the course of life and already during the first year of life the round, ball-shaped form of the infant body approximates more and more the elongated, almost cylindrical form of the adult body. With the transition from ball to cylinder, however, the surface diminishes in relation to its mass. The change in body shape and the steadily changing relationship between body surface and body mass explain why the caloric requirements, calculated per kilogram, decrease from month to month and from year to year. If the body surface would be chosen as basis for calculating the nutritional requirements, they would in a resting individual remain approximately the same regardless of age. Or, more generally stated, according to the surface law of *Rubner*, "the energy consumption in the fasting, resting mammal of different size is in proportion to the body surface."

Serious objections have been raised against this theory by *v. Pfaundler*. In its original form by *Rubner* the law applies only to fasting animals. The progress in deterioration of the nutritional state and the resulting changes of body surface in a period of hunger must lead to a reduction of energy consumption in the different periods of starvation. Furthermore, the effects of voluntary and involuntary motions which influence food metabolism considerably, are not taken into consideration. The condition of the body surface (e.g., hairy—not hairy, protected—unprotected) also changes the amount of heat loss. And finally the changing atmospheric influences can act in a restricting or promoting way on heat loss from the surface. For the resting infant differences in metabolism up to 100% have in fact been observed so that somewhat exact averages for calculating nutritional requirements, even on the basis of body surface, can hardly be established. Calculation of nutritional requirements on the basis of body surface would only then become more exact if apart from external body surface the internal body surfaces (intestines, lungs) could at least partially be taken into consideration since their total surface is by far larger than the skin surface of the body which so far has only been considered. A calculation of the size of the internal body areas is, however, practically impossible.

> *V. Pirquet* attempted to introduce in place of calories the caloric value of NEM (Nahrungs Einheit Milk–1g milk = 0.67 calories) for calculating the nutritional requirements. However, the application of the so-called Nem-system, which bases its calculations on the size of the absorbing intestinal area, has not proven more exact than the older caloric calculations because fundamentally it is also based on the caloric system. The size of the intestinal surface illustrates perhaps better than body surface or body weight the close relationship between food intake and food metabolism. The extent of intestinal surface corresponds approximately to the square of the sitting height of the child, that is, the distance between crown and os ischii.

Despite all objections and doubts the calculation of the nutritional requirements on the basis of body weight or body surface must for practical purposes be considered the most usable measure. The calculation of nutritional quantities must therefore be made with the understanding that despite all figures, formulas and rules we do not deal with a mathematical law but with a vital function which is merely estimated on the basis of an empirically arrived at rule. In the majority of children, therefore, we can in this manner arrive at only approximate indications regarding an adequate total quantity of nutritional substances.

Utilization of Ingested Food

The food intake of the infant has to fulfill the following tasks:

1) *Requirements for basal metabolism*, which is a rather constant quantity.
2) *Requirements for growth processes*, which vary greatly in the different periods of the first year.
3) *Requirements for muscle work, crying* (including digestive work).
4) Compensation of caloric losses through *elimination*.

Basal metabolism at birth amounts to about 40 to 50 calories per kilogram of body weight, increases steadily and reaches a maximum of about 55 to 58 calories per kilogram of body weight towards the end of the first year of life; from then on it decreases again gradually during the course of the following years.

The amounts required for growth processes (increase of protoplasm, fat, connective tissue) which is clinically expressed by increase in length and weight are about 50 calories per kilogram of body weight in the first period of life. With a slowing down of the intensity of growth processes this part of nutritional requirements decreases very rapidly. At the end of the first year of life it amounts to only 20 to 25 calories per kilogram of body weight.

The values covering the losses due to muscle activity (restlessness, crying, etc.) vary considerably. Approximately 6% of the caloric intake is utilized for digestive processes. The motions of a resting infant take up about 10 calories per kilogram of body weight.

A further additional factor is a requirement of about another 10 calories per kilogram of body weight (that is, 10% of caloric intake) for losses of calories in the urine, stools etc. We therefore find:

Caloric Consumption for:	*Infant During First Months*	*Infant at 12 Months*
Basal Metabolism	40–45	55
Growth	50	25
Muscle Activity	10–15	10
Loss through Elimination	10	10
	100–120 calories	100 calories

The different nutritive substances do not participate in equal measure in the total metabolism of the body. Experience in breast fed infants shows that about 1.8g protein per kilogram of body weight per day are required for normal development of the child. In the artificially fed child the protein quantities required are estimated somewhat higher, at 2g, perhaps up to 3g per kilogram of body weight per day. However, not only the quantity but also the quality of the protein is of significance. The healthy infant requires about 7 to 9g of sugar per kilogram of body weight per day. An accurate estimate of carbohydrate requirements in the artificially fed child is hardly possible, especially if the food is mixed (vegetables, fruit), since the quantities of carbohydrates contained in these foodstuffs vary greatly and are not utilized equally in the intestinal tract. A daily quantity of about 10g sugar per kilogram of body weight is probably adequate for the majority of infants for covering the carbohydrate requirements. Similarly the daily fat requirements can only be estimated approximately. Six to 7g per kilogram of body weight per day are considered adequate for the infant. In general it is estimated that during infancy 40% of the caloric intake should be in form of carbohydrates, 50% in fats and 10% in protein. To this come water, salts and vitamins.

The water requirements of the healthy infant are very great compared to those of the adult. This is indicated already by the high water content of the body of the child and the large amounts of water contained in natural and artificial food mixtures:

1 liter breast milk contains 885g water
1 liter cow's milk contains 874g water

Calculated per unit of body weight, the water intake of the healthy infant amounts to about six to eight times the requirements of the adult. In the infant the amount averages 140 to 150g per kilogram of body weight. It is highest at the end of the first month of life after which it decreases gradually.

The average of 150g water per kilogram of body weight represents approximately the optimum for development. However, experiences in feeding young infants with high caloric, concentrated food mixtures show that development is possible in a large number of infants even with a lower water intake (100 to 120g per kilogram of body weight). A water deficient intake is undoubtedly unfavorable for the infant. Thus we see that a calorically adequate but water deficient diet results in a lack of weight gain. This stoppage in weight gain can be improved solely by increasing the water intake, without any other changes in diet (see Chapter on Growth). Such disturbances are found most frequently in prematures and in infants during the first three months of life, when water requirements are particularly high. Beyond that, however, larger water deficiencies in the food can lead to general, serious disturbances of the metabolism, to fever, diarrhea, etc. (see later: Damages due to deficient water intake).

The mineral requirements are adequately satisfied by breast milk whey and even more by cow's milk whey which contains large quantities of salt. Only calcium and iron form an exception in some children. If the older infant does not take in iron in adequate quantities in form of vegetables and fruit or if calcium is no longer present in a form which can be biologically utilized by the organism due to exceptional metabolic conditions (see chapter on tetany, rickets) the results might be an iron or calcium deficiency.

To indicate absolute figures for salt requirements is the more difficult since the requirements for the different salts vary greatly depending on processes in the intestinal tract and variations in the amount of other food substances (for instance the relationship of calcium to fat). The quantities of salt contained in milk whey and in the mixed diet of the infant are more than adequate so that disturbances due to salt deficiencies hardly ever occur in a healthy well fed child.

Not all vitamin carrying food substances contain the same amount of vitamins. The same sorts of fruit or vegetable will, for instance, show changes in their vitamin C contents due to differences in radiation, fertilization, etc. Winter milk is in every respect poorer in vitamins than summer milk. Cod liver oil shows variations in its vitamin D content according to origin and method of preparation. In addition, vitamin requirements of the individual infants are subject to strong variations on the basis of differences in constitution and condition as well as age. In the healthy, developing child the vitamin requirements seem to be relatively low. However, any pathological disturbance, rise in temperature, diarrhea, etc. immediately raise the vitamin requirements considerably. In general it can be stated that in a balanced diet the requirements for vitamins A and B are covered. In order to supply the vitamin C requirements it is necessary after the third or fourth month of life to add foodstuffs containing vitamin C. The following table applies to the healthy infant:

		Contained in	
	Quantity Required Per Day	*1 Liter Cow's Milk**	*1 Liter Breast Milk*
Vitamin A	1500 I.U.	300–750 I.U.*	1500–1800 I.U.
Thiamin	0.4 mg	0.45 mg (0.3–0.7 mg)	0.3–0.6 mg
Riboflavin	0.6 mg	2 mg*	2–3 mg
Nicotinic acid	—4 mg*	0.7 mg*	
Pantothenic acid	—5 mg*	3 mg*	
Pyridoxine	—2 mg*	2 mg*	
Vitamin C	30–40 mg	5–20 mg	40–70 mg
Vitamin D	400–800 I.U.	20–30 I.U.	Traces

* No definite figures can be given since they vary considerably with the type of fodder of the milk supplying cows.

CHAPTER III

Methods of Infant Nutrition

BREAST FEEDING

The need of nursing, that is, feeding a newborn or an infant according to the principles of nature, must be regarded even today as fundamental requirement despite all progress made in artificial feeding. The problem of natural feeding is not settled by proclaiming in words and in writing that it is the noblest duty of the mother to nurse her child, a duty which no woman should shirk. These and similar phrases assume after all only one prerequisite for the fulfillment of the nursing process: they demand the desire on the part of the mother to nurse her infant. In practice the fulfillment of this demand is not always such a simple task either for the physician who supervises breast feeding or for the mother who carries it out. Even the much repeated phrase that almost every mother can nurse her child is not absolutely correct. It applies best to mothers who deliver their children in the atmosphere of an obstetrical station. In the first eight to 10 days following delivery almost every mother can produce the relatively small quantities of milk which are necessary for feeding a newborn. It is thus understandable that obstetricians will attribute to 90–100% of all mothers the ability to nurse their babies.

It is, however, an entirely different problem to carry out breast feeding if the mother lives in her own family circle especially if in addition to socially and hygienically unfavorable surroundings the mother and her closer environment show a higher degree of sensitivity and intelligence. In these women certain feelings of inadequacy regarding their nursing ability are accompanied by fear whether the child receives enough food as well as by worry which so frequently originates from the opinions of outsiders. In this manner it is easy to create an atmosphere which is far from conducive for the smooth functioning of nursing. Such circumstances help to explain the fact that satisfactory nursing, that is, breast feeding for at least three to four months, is possible only in about 70% of mothers, especially in the middle classes, intellectuals, etc. About a third of the remaining 30% are able to partially nurse their babies for some months, while the other two thirds are for some reason completely unable to produce adequate milk secretion or after a hopeful start milk secretion stops altogether within a few days or weeks after birth. These mothers, and particularly primiparae, who despite their desire to nurse are unable to produce milk, are subject to a kind of fear neurosis. These nervous disturbances have been described as "maternity neuroses." The essential cause for this neurosis is the tremendous overrating of any expression on the part of the infant, showing the slightest deviation from what mother and relatives consider "normal." In addition there are feelings of inadequacy which develop, again mainly in responsible mothers of first children, if the milk flow in the beginning is not as ample as the mother considers desirable. The fear not to be able to nurse results not infrequently in the actual inability to breast feed the child. The physician must be prepared for such neuroses and one of his main tasks in the supervision of nursing is to fight against them, especially in more intelligent mothers. Only if the mother can be calmed and convinced of her ability to nurse is it possible to carry out breast feeding.

Apart from fear of not having sufficient milk the neurosis of young nursing mothers manifests itself in an exaggerated observation of the child. Stools are carefully viewed and evaluated as to number and appearance. Any regurgitation upsets the mother. Physiologic changes of the skin of the newborn or any minor soreness, the frequent skin rashes on the cheeks, give rise to undue worry. The weight of every meal and weight progress from day to day is controlled in greatest detail and any deviation from the expected, almost clock-work regularity of weight increase causes new fears. The numerous text books of infant care, some of good, some of doubtful quality, are studiously read during those weeks and on the basis of these books the "normal curve of development" is overrated and any deviation in either direction gives rise to new worries and fears in the mother.

Furthermore, there are widely spread and always unfounded doubts as to the quality of the milk which may be considered either too fat, too thick or too thin. All these doubts and observations, together with certain difficulties and complaints which develop in the process of nursing such as low back pain or pains in the breast etc., lead to a constant fear of the next meal which in the opinion of the mother will reveal new inadequacies in her nursing ability or new abnormalities of the child. Any good judgment is lost. Thus there develops a readiness to willingly follow just any advice. Finally calmness and confidence, which are so necessary for the normal process of nursing, are replaced by strong restlessness on the part of the mother which is in turn reflected upon the child which now begins to cause real difficulties, by mistakes in the technique of nursing and by instinctively feeling the restlessness of the mother. This influence of psychological processes on the development of nursing explains why phlegmatic women frequently make the best feeders while sensitive women, wives of physicians or women physicians or nurses who become mothers and who know too much about infant feeding often fail in their efforts to nurse.

The most important task of the physician in all these cases is to awaken and strengthen self-confidence in the doubting mother and furthermore take care that calmness and confidence pervade the surroundings. The physician can reach this goal only if he can correctly size up and evaluate the minor disturbances and difficulties which threaten the continuation of nursing. The undisturbed, school-like process of nursing is described only in books. In practice it is extremely rare to go through this period without any difficulties on the part of mother or child. The attitude of mother and child toward each other is in practice always subject to certain difficulties. The joy of nursing therefore develops in many mothers only after the critical initial stage has been overcome. The healthy development of the child is the most important factor contributing to the attainment of this harmonious condition; it is the best sedative and the best galactagogue for the mother. The feeling of insufficiency and the worries of the mother disappear more readily in the face of satisfactory development of the child than through any written or spoken instructions. It is therefore an essential part of the physician's task to see that the child gains in weight. For the weight gain is for the mother the criterion which confirms her ability to nurse. This maternal expectation is often not fulfilled due to too strict, prolonged continuation of exclusive breast feeding at any cost. By occasional addition of small amounts of artificial food this goal can usually be reached and for psychological reasons this procedure is frequently unavoidable. At times this is the only way to persuade the mother to continue nursing the baby.

It seems that in practice breast feeding encounters a number of difficulties which are more frequently on a psychological than physical basis. The basic technique of breast feeding is today known in all lay circles, thanks to the enlightening efforts of mother and child clinics as well as to the popular literature. Errors in this direction are hardly ever made today. The rooming-in system, whereby the mother participates from the first day of life, while still in the obstetrical department, in the care of the child, is no doubt of advantage to the mother in obtaining instructions in nursing technique and of help in overcoming initial nursing difficulties. However, it seems indicated in the interest of the mother to start this rooming-in arrangement only three or four days after birth. One of the disadvantages of the system is the fact that the newborn is more exposed to infections, if either several mothers share a room in a hospital or if the mother, occupying a single room, receives many visitors following her confinement.

In any event it should first be attempted to establish a nursing procedure along generally accepted principles. Five meals a day have so far been considered normal. Individual physicians and in some countries more frequent nursing is still advocated, especially in the first days of life, with up to six, or eight or even ten feedings a day. But only with five meals is it possible to obtain a longer rest at night which offers mother and child the necessary period of sleep.

Recently an arrangement of the feeding schedule according to the wishes of the infant has been recommended from several quarters (self-regulating feeding program or self-demand schedule of infant feeding). According to this system the mother may and should nurse the baby every time he expresses by crying and by restlessness the wish to drink. We are thus about to return to feeding methods which were still customary 50 or 60 years ago and which among primitive people still exist today. The essential reason for this system, which in fact represents a return to nature, is given as the desire to avoid feelings of discomfort in the newborn. After already a short period, sometimes even after 8 to 14 days, most infants demand only five or six meals a day, that is, they themselves adjust to the schedule which has so far been customary. This system, however, has certain disadvantages for the mother, especially during the first days after birth when she requires much rest. In addition, this self-regulation should be carried out in infants receiving only breast milk. All artificially fed infants remain on the old system of five or six regulated meals a day, in order to avoid damages (overfeeding). So far we have absolutely no proof that the more frequent crying and whining of infants whose schedule during the first days of life is regulated by adults in any way causes psychic damage for their later life. Except for those infants where crying and yelling should be avoided for reasons of health it seems, therefore, not absolutely indicated to abandon the system of purposeful regulation of five or six meals per day and to establish in its place a system of self-regulation whereby the feedings take place according to undefinable desires of the child.

In order to carry this system out correctly a certain amount of intelligence and understanding, mainly on the part of the mother and even on the part of the father must be assumed. It has therefore rightly been emphasized that this system of infant feeding can be carried out in only a small part of the population. The suggested compromise of a semi-self-demand feeding schedule (*Glaser*) therefore seems more acceptable. This is a method which in similar form has probably been practiced all the time by nursing mothers, even without consulting the physician. Within the frame of a fixed schedule of meals the nursing mother may, under this system, make her own changes if she concludes from the behavior of her child that satisfaction of the infant's appetite is desirable.

Under the system of five meals a day, which is instituted

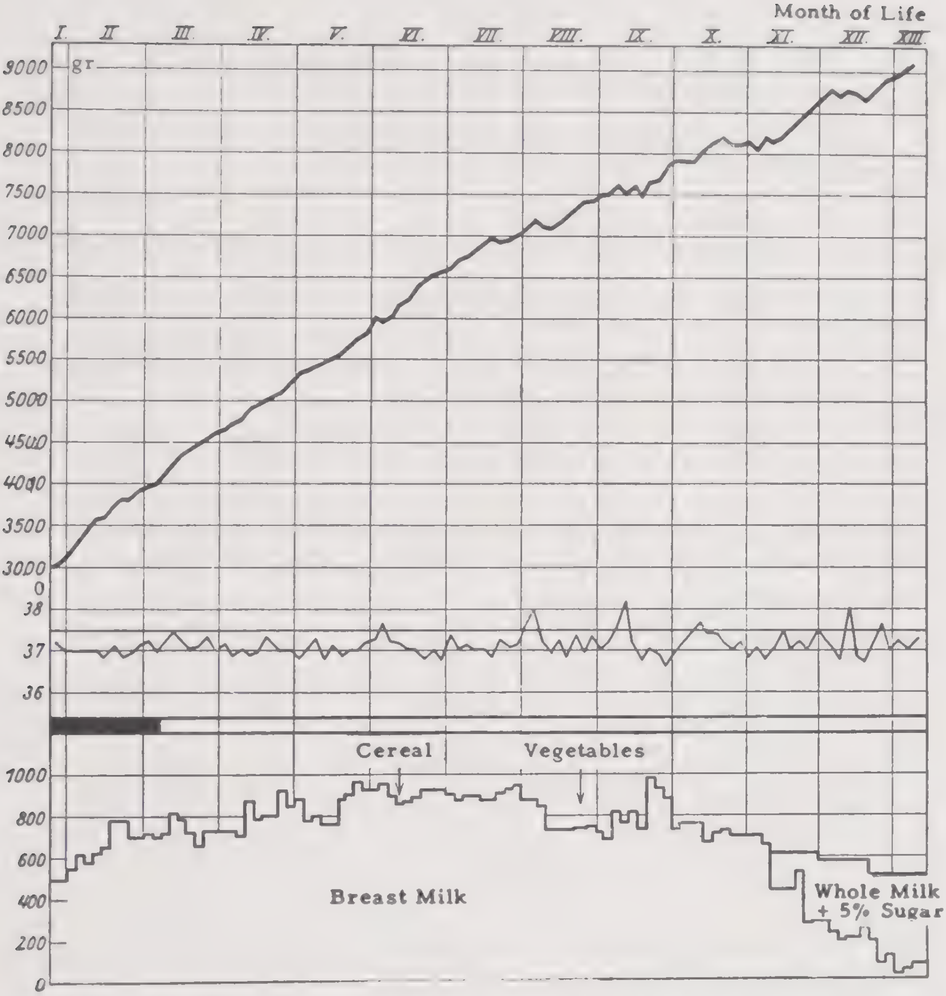


Fig. 10. Normal development of an eutrophic infant with breast feeding. *Note:* In this and in all following graphs ■ represents bad stools and ☐ represents good stools.

from the beginning or after a period during which a greater number of feedings was given, it is customary to offer the first meals in the early hours of the morning, between 6 and 7 o'clock, and then nurse the child in regular intervals of four hours. The duration of a single feeding should not exceed 15 or 20 minutes at the most. It has repeatedly been established that the infant takes by far the greatest part of the meal within the first five minutes while after 15 minutes he takes only a few swallows, giving him very insignificant amounts of milk. The unlimited extension of feedings which young mothers favor in the hope of giving the child still more milk, brings no advantages to either mother or child. The mother who has hardly time to rest between the prolonged feedings feels the nursing process as a burden. In addition there is a danger that the nipples might become sore through the continuous irritation of sucking. Putting the child on the breast causes pain and the fissures and rhagades of the nipple become portals of entry for infections. This can lead to mastitis.

The infant empties the breast through chewing motions (see p. 68).

> In the excretory ducts of the breast are enlargements (milk sinuses) the position of which corresponds about to the pigmented areola. When nursing the infant grasps the areola hermetically with his lips. His opening of the mouth causes a reduction of pressure in the system breast ducts–milk sinus–oral cavity which results in a flow of milk from the glands into the milk sinus. Upon closing the jaws the pressure of the lips causes the emptying of the milk from the milk sinuses through the excretory ducts which terminate in the nipples, into the mouth of the child.

This technique makes it understandable that the infant can suck, swallow and breathe simultaneously when taking nourishment. These facts are important for the technique of nursing. They prove that it is not the form of the nipple which is decisive for the ability of the mother to nurse. Flat and even moderately depressed nipples do not prevent nursing. Mothers must be instructed that the infant must grasp not only the tip of the nipple. It is important that part of the areola is grasped by the lips. In small, underweight children a very large and strongly erectible nipple might occasionally cause difficulties in nursing and even at times make sucking impossible by causing retching and vomiting of the infant.

The quantities of milk which the child with normal sucking ability and with adequate breast secretion takes in, increases in the course of two to three weeks to the quantity of 600 to 700g, corresponding to the body weight of the child. The average total milk intake can be calculated according to a well known rule as follows:

(Age in days n–1) × 70–80g
for example: on the fourth day: (4–1) × 70–80g = 210–240g
on the tenth day: (10–1) × 70–80g = 630–720g

Deviations from these figures in either direction are frequent and without significance provided the child gains weight after the period of physiologic weight loss.

The size of the different feedings varies considerably in the course of the day, depending partly on the intensity of milk secretion in the breast, partly on the sucking desire of the infant. Usually the first and second meals of the day are the largest, the third feeding around noon is usually smaller, the afternoon feeding frequently large again while the last feeding in the early hours of the night is again smaller, especially in somewhat older infants since they are frequently asleep at that time and it is difficult to awaken them. All these amounts, the total daily quantity and the amounts of single feedings vary from one child to the other within wide limits. From none of these figures is it possible to form an opinion whether additional food is necessary or whether it is worth while to continue nursing altogether. Only in connection with a control of the body weight is it possible to judge whether milk secretion is adequate or not. Because of these changes in quantities taken during single feedings caution is advisable in drawing conclusions from the size of one individual meal on the actual quantities taken in a day as well as on the nursing capacity of the mother. The necessity for additional food can be established only by weighing all the meals or at least several meals a day. If the child is weighed weekly and shows a gain of at least 150g the quantity of the milk intake should be regarded as adequate even if it is considerably lower than the expected intake calculated on the basis of the child's weight.

Around the tenth day of life milk flow should be sufficient to cover the nutritional requirements. The undernourishment of the first few days is of no significance for the child. If the milk flow begins around the third or fourth day after birth, frequently even later, it takes about another eight days until the breast functions fully. During the days of insufficient food intake the child should be given additional small quantities of weak tea, sweetened with saccharine—about 50 to 200g per day, in order to cover the fluid requirements of the infant and avoid thirst. The amount of the so-called physiologic weight loss is also greater if the fluid intake is inadequate.

> Beginning signs of thirst are dry skin and subfebrile temperatures. If the water deficiency is severe prolonged dangerous manifestations may develop. The temperature rises, the skin is hot and dry, frequently very red or cyanotic. The urine contains leukocytes (pyuria of dehydration). The blood thickens, as evidenced by a high hematocrit value. The dehydration of the newborn can finally simulate the picture of a severe septic illness. Adequate water intake causes all pathological symptoms to disappear within a few hours (see also p. 251).

The period of inactive waiting in case of insufficient milk secretion and lack of development of the child should be limited in time. Beginning with the third week of life all children who do not at all or not adequately gain in weight must receive sufficient nourishment to cover their needs, either by changing the nursing technique or by adding artificial food. Prolonged hunger causes the same unfavorable effects in the breast fed child as in the bottle fed child. Dystrophic conditions, accompanied by a number of disagreeable secondary symptoms develop also in the hungry breast fed infant although they might develop more slowly than in the bottle fed infant. Treatments which increase the milk secretion, such as medicines (hormones) or irradiation (e.g., ultra-violet rays) have no effect. The favorable results which might be obtained by these procedures should be considered on a psychological basis and in this sense could be used to advantage. Even the manual expression of milk after the child is taken from the breast does not increase the amounts of milk produced by the glands.

The physician taking care of the child must also supervise the mode of living of the nursing mother. Most women do too much rather than too little for the care of the breast. Massage, washing, expression, etc. of the breast during pregnancy are unnecessary. Before and after delivery and throughout the period of lactation it is sufficient to wash the nipples carefully with cool water. Massaging of the breasts and washing with so-called disinfectants such as boric acid or alcohol are superfluous or even harmful since they cause dryness and brittleness of the areola and thus promote the formation of rhagades. Putting small amounts of pure vaseline on nipple and areola after every feeding is preferable to washing. Before the next feeding the remaining vaseline is removed with a soft dampened linen cloth or with cotton. In order to avoid the formation of fissures the child should be put on the breast in such a manner that the nipple hangs into the mouth of the child without tension. If the nipple is pulled upwards the formation of rhagades on the lower part of the nipple is hardly avoidable.

Already during pregnancy an adequate diet of the mother is of utmost importance. Adequate nutrition during pregnancy should improve chances of a normal delivery for mother and child. The number of premature births and underweight children is larger in women who had an inadequate diet during the second half of their pregnancy than in women who ate a complete diet. The nursing mother should be weighed regularly about once a month. If the mother loses weight a physical examination of the mother is advisable, possibly also a change in nutrition of the child (e.g., partial weaning). Hardly any restrictions are imposed today on the mother's own diet. The plentiful intake of rich cream soups, flour soups and of large quantities of fluids which mothers are at times advised to take are of no avail since this type of nutrition does not increase the milk supply and is carried out by many nursing mothers only with great aversion and frequently it takes away the pleasure of nursing. Any superfluous food intake or forced feeding only tends to increase the tendency to adipositas which is present in many nursing mothers. Fattening is also undesirable because milk secretion seems to diminish if the mother puts on fat. Any special rules for the diet of the nursing mother are superfluous because no known food will have either a favorable or an unfavorable influence on milk secretion. It has not been proven whether certain types of food—cabbages were primarily suspected—bring about a change in the milk which makes it less desirable for the child. If, however, a mother believes that a certain type of food might have such unfavorable effects on the condition of her child it might be advisable for her to abstain from cabbage, salad, raw fruit, or whatever else she believes to be the cause. The physician will permit the nursing mother to eat everything. In order to cover the additional food requirements which are caused by supplying the child daily with $^3/_4$ to 1 liter of milk, about 500 to 700 calories and almost a liter of water, it is necessary to compensate for these additional losses by approximately equal additional intake. Since apart from the calories contained in the secreted milk the nursing breast expends considerable labor in secreting the milk it is not sufficient to add to the usual diet about the same amount of milk as the mother supplies to the infant. According to a simple rule the nursing mother should eat about 50% more than she did before she started to nurse. The type of food with which she covers these additional requirements seems to be quite immaterial. However, the fact that the mother furnishes about $^3/_4$ to 1 liter of water in the breast milk causes a feeling of thirst which makes it easy for her to drink one liter whole milk per day. Frequently this milk is enjoyed even by those women who usually do not care for this food. The milk also furnishes the amounts of protein and calcium which the child requires for the formation of cells. If the mother shows an aversion against milk the physician should accede to her wishes regarding diet. An adequate protein intake in form of meat or cheese will prevent a protein deficiency in the organism of the mother.

Under no circumstances would the type of food of the mother influence the chemical composition of the milk. Regardless of diet the composition of the milk remains unchanged, showing only individual variations within narrow limits. The minor variations in the composition of the milk which occur in the course of lactation and which have at one time been proven in complicated, troublesome investigations are without practical significance. Even severe restrictions in the mother's diet in quantity and composition, as were experienced for instance during the war years, had no essential influence on the composition of the milk. Deficiencies in the development of the infant were not observed even if the mother suffered considerably from the results of malnutrition and the requirements of nursing.

The problem as to the influences, if any, of the re-appearance of menstruation in the mother on quantity and quality of milk produced during those days is as yet not finally settled. While some authors deny that there is any connection between menstruation and disturbances in the nursing procedure, other authors acknowledge such relationships. The existence of so-called Menotoxins in the milk of menstruating women which *Schick* tried to prove by showing that flowers would wither more rapidly in the milk of menstruating women than in the milk secreted at other times, has been contradicted. The assumption that the reduced intake of breast milk during the days of the mother's menstruation is not the result but the cause of the menstruation, which sets in as soon as milk secretion in the breast decreases, probably applies only to occasional cases. However, there are a number of children who take in less food during the time of menses and increase their food intake again to normal after a few days when menstruation has ceased. Changes in the taste of the milk during the days of menstruation might be the cause since infants are very sensitive to any changes in taste and therefore do not care for the milk during those days. Whether or not menstruation influences the nursing process might also depend on the extent to which the general condition of the mother is affected during that time. In this we find considerable variations among women. In practice these disturbances would hardly reach a degree which would necessitate changes in nursing technique or even weaning.

The influence of psychological upsets on quality and quantity of milk is also rather doubtful. The possibility of a change in the composition of the milk due to worry, anger, fright, etc.—which formerly were favorite weapons used by wet nurses in the homes—has not been proven. The experiences of the war years during which many women nursed their children under the burden of great worry and distress did not contribute to prove this assumption. It must be admitted, however, that in sensitive women sudden great excitement or fright might cause a temporary stoppage in the secretion of the milk glands, as of all the other glands of the human body.

A new pregnancy also need not in itself be an obstacle in the continuation of nursing. It is, however, in the interest of the mother not to continue nursing beyond the third or fourth month of the new pregnancy. The nursing infant as well as the growing fetus demand their shares of the mother's strength. It is therefore advisable to start weaning the child around this time.

The quantities of even the most powerful drugs which are transmitted through breast milk are so negligible that they cause no harmful effects in the child. This particularly applies to the narcotics and sedatives of the different types of the morphine and barbiturate group which are frequently feared but which, if taken by the mother in the customary dosages, cause in the child a temporary sleepiness at the most. There should be no hesitations about the mother taking arsenic preparations (antiluetica), sulfonamide preparations and antibiotica since these drugs are transmitted in the milk in such small quantities that they would not even have a curative effect on the child. Even a general anesthesia of the mother is tolerated by the child without harmful results, although it is understood that the child should not be put on the breast until the effect of the anesthetic has worn off. Women used to excessive smoking should limit their consumption of cigarettes. A consumption of more than six to eight cigarettes a day can lead to a transmission of nicotine in the milk in such quantities that the infant develops the first signs of nicotine poisoning (nausea, diarrhea).

In an adequately functioning breast the secreted milk quantities correspond to the requirements of the infant. If a child receives only breast milk the amount of the secretion varies betwen 3/4 to 1 liter per day, once nursing has been established. At times the quantities of milk are somewhat smaller with, however, a higher fat content of the milk amounting to as much as 6, 8 or up to 10%. If two infants are nursed, as in the case of twin birth, the healthy and strong mother can supply double quantities of milk for even a number of months. The average performance of the milk glands can be increased considerably under special demands or in mothers with special psychological or physical constitution. Among the wet nurses in institutions who formerly supplied several infants with breast milk or who produced larger quantities of milk by manual expression, quantities of 3 to 4 liters per day over a longer period of time were by no means rare. The record is probably held by a wet nurse of an infant home who furnished more than 16 hectoliter milk during the 51 weeks of her activity as wet nurse, of which 6.9 hectoliter were manually expressed and 9.3 hectoliter were taken by the infants from the breast. The maximal daily secretion amounted here to almost 7½ liter. Although such quantities are exceptional they are nevertheless instructive examples how adequate stimulation can increase the output of a gland.

The period of lactation apparently can also be prolonged considerably beyond the usual term of six to eight months, as experiences in the folklore have shown. A spontaneous drying up of the milk glands does not occur so easily once the milk secretion begins to function. The desire to stop nursing and the now recognized necessity to add other foods after a certain age even in the breast fed child are more apt to terminate the milk secretion before the physiological function ceases. The increase in milk quantities as well as the continuation of milk secretion beyond the customary period are, however, possible only if the constitutional predisposition is accompanied by the desire to continue nursing. Nursing ability is probably not hereditary. A prediction regarding the expected output of the breast is hardly possible in the individual case. The ability or inability to produce sufficient milk changes considerably in different children of the same mother, so that the mother might be able to nurse one child adequately and another child not at all, without any apparent cause for these variations. In other women a short period of satisfactorily increasing milk secretion after each birth is followed regularly by a drying up of the breast after a certain length of time.

Size and form of the breast, strong or weak pigmentation of the areola, abundance or scarcity of palpable parenchyma, emptying of the milk through pressure in a stream or only in drops, vascularity or pallor of the skin of the breast and many other indications do not permit any judgment regarding the expected performance of the breast. The secretion of milk from the breast between feedings, which soaks through the mother's clothes, is frequently observed in women whose milk secretion ceases early. If this milk secretion continues for some time, the comparison of the temperature of the breast fold with that measured in the armpit permits an approximate judgment regarding the functioning of the gland. A difference in temperature between breast fold and armpit of more than $^{1}/_{2}^{\circ}$ in favor of the breast points toward satisfactory activity of the breast. Equal temperatures or smaller differences in temperature are observed in cases where the functioning of the breast is not adequate.

Every woman should attempt to nurse her child even if the output of the breast is small. There are very few contra-indications against this demand from the physician's point of view, although it must be admitted that the request for breast feeding is frequently made more theoretically than can be carried out in practice. An absolute indication against nursing is active or recent tuberculosis of the mother. The dangers of tuberculous infection are greater for the infant than the dangers of a well planned artificial feeding. Even the immunizing of the infant with BCG immediately after birth does not reduce this danger since "immunity" develops only in the course of several weeks. Under the strain of nursing any recent tuberculous infection of the mother can easily be activated again. This endangers the child, even in later months of life. Contra-indications for nursing are also other severe diseases such as nephritis, true diabetes and decompensated heart disease. Lactosuria, which occurs frequently in pregnant and nursing mothers and is always harmless is, however, no contra-indication. Severe psychoses which affect the physical condition of the mother or cause unreasonable excitement disturbing the sense of responsibility of the nursing mother make a weaning of the child imperative. This applies also to epilepsy where a sudden loss of consciousness during an attack can endanger the child during nursing. Breast feeding also seems to have an unfavorable effect on mothers suffering from Basedow's disease. Severe typhoid fever or severe dysentery make a discontinuation of breast feeding necessary because of the danger of transmitting the disease to the infant. If the mother develops scarlet fever, diphtheria or measles a temporary interruption in nursing is indicated even under the assumption that most infants are immune against these diseases. These assumptions, however, do not apply to all children. In cases of erysipelas and prolonged puerperal fever of the mother breast feeding should also be discontinued, since a transmission of the infection to the child cannot always be avoided even under most careful observation of an isolation regime. The decision to discontinue nursing is made easier by the fact that milk secretion decreases considerably with the development of any disease connected with high temperatures in the mother.

Lues in the mother is no contra-indication against nursing. If a mother suffering from syphilis during the last months of pregnancy is given radical treatment it is quite probable that a transmission of the disease to the infant can be avoided. In those cases where adequate preventive treatment in the mother was omitted and the child was most likely infected before and during birth, breast feeding will increase the resistance of the child and might even reduce the manifestations of the disease through the intake of antibodies through the milk. The few children—about 5%—who despite a serologically still positive but asymptomatic syphilis of the mother remain clinically and serologically free from infection, that is, in whom infection did not occur, can without doubt according to present day knowledge be nursed on the breast despite the latent syphilis of the mother, at least during the first few months. It seems advisable, however, to wean those children around the fourth month of life.

Special consideration must be given to children of diabetic mothers, especially mothers whose diabetes was poorly regulated and who had high blood sugar during and at the end of their pregnancy. The number of such newborns increases steadily since more and more of these mothers reach child-bearing age. These newborns are almost always exceptionally large. The heart is hypertrophic. The rate of mortality of the children during and immediately following birth is high especially if there are also malformations which occur not infrequently. The blood sugar which post partum is the same in mother and child, decreases in the newborn within the first hours of life (similar to that of children of healthy mothers) to low values of 40 to 75 mg% and becomes more or less constant around the second to fourth day of life. The cause of the high mortality of children of diabetic mothers is not yet sufficiently known. Probably the disturbances in the harmony of internal secretion are the cause for the lability of these newborns. The children are frequently very fat and edematous at birth. Enlargement of liver and heart are noticeable. Collapse in the first hours after birth, asphyxia, convulsions and cyanotic discoloration of the skin which resembles more the red skin existing in diabetics, and atelectasis are dangerous complications in the first days of life. By giving oxygen or sugar orally within the first hours post partum part of these dangerous symptoms will disappear. For prophylaxis an early regulation of the diabetes in the pregnant woman, especially a lowering of the high blood sugar values at the time of birth is necessary. Occasionally a premature termination of pregnancy is advised.

In practice the most frequent disturbance in the nursing process is the appearance of inflammatory symptoms on the breast glands. If mastitis or rhagades on the nipple develop, the question is raised whether a continuation of nursing is advisable. The question is not always solved by stating categorically that rhagades and inflammations are no obstacles in the continuation of breast feeding. Those who have witnessed how even less sensitive women have cried out in pain when putting the child on a fissured nipple will not lightly give the advice to continue nursing. If the previously mentioned measures in nursing technique and the adequate care of the nipple fail to avoid the development of fissures it must be attempted to maintain the natural food for the infant by changes in the nursing technique and treatment of the rhagades. Sucking through a breast shield is usually unsuccessful; it frequently causes the infant such difficulties that the quantities of milk rapidly decrease and the incomplete emptying of the breast causes congestion of the milk and subsequent drying up of the breast. The rhagades themselves should not be treated with a silver nitrate stick. It is sufficient to apply vaseline or a 1% argentum nitricum—10% balsam of Peru salve or similar preparations which are carefully removed before the next feeding. Healing of the rhagades can be accomplished most rapidly if the child is not placed on the affected breast (or the more painful breast, if both are affected) for one to two days. If the breast becomes too full the mother can carefully express the excess of milk by hand. After two days the rhagades are usually sufficiently healed to permit a resumption of nursing.

During the sucking of the child the rhagades at times start bleeding. The blood is swallowed by the infant and frequently causes bloody vomiting or the blood reappears in the stool (pseudo-melaena), which then assumes a dark-brown or black color. The discovery of bleeding fissures on the nipple will occasionally avoid a wrong diagnosis (true melaena, intussusception, intestinal ulcers etc.).

The use of so-called breast shields made of lead should be avoided. Some women believe that by wearing these protective shields they can avoid the development of rhagades. The small quantities of lead from these shields which remain on the nipple are sufficient to cause in some children severe lead poisoning which develops very slowly and appears sometimes still during the period of nursing, but frequently only weeks and months after termination of breast feeding in form of vomiting, lack of appetite, anemia, high temperatures and the symptoms of severe, even lethal encephalitis. The diagnosis can be definitely established through the finding of stippled erythrocytes, the roentgenological proof of lead lines on the epiphyseal ends of the long bones and the sutures of the skull bone, and possibly the presence of lead in the urine. It is doubtful whether the treatment with B.A.L. is of advantage, particularly when the diagnosis of lead poisoning is made very late. The mobilization of lead from the bones, which can then damage the central nervous system, is occasionally considered a contra-indication for the use of B.A.L.

The main danger of rhagades lies in the fact that they can become portals of entry for infections and thus precursors of a mastitis. The mastitis which takes place in the interstitial tissues should be differentiated from those inflammations where the infection spreads in the parenchyma of the gland. Parenchymatous mastitis can be traced to congestive processes in the breast which are secondarily infected from the milk ducts. The general symptoms—pain, fever and poor general condition—can be of considerable severity. While it is possible to advocate continuation of nursing as long as the gland shows no abscessed sensitive nodules, the child should not be put on the diseased breast if pains are severe and an abscess is forming. In case of *interstitial mastitis* which begins with high temperatures, chills, glandular swellings and the symptoms of lymphangitis, nursing should be discontinued in the interest of the mother. Through energetic penicillin treatment, given as early as possible, it is today almost always possible to heal the developing mastitis so that nursing can frequently be resumed within a few days. If treatment was not started sufficiently early and an abscess developed in the breast, nursing must be discontinued also in the interest of the child. Experience shows that despite most painstaking cleanliness abscesses in the breast are followed by skin infections in the child. Recurring abscesses, erysipelas, osteomyelitis and other septic diseases originate in the infant in this manner. If in case of suppurative mastitis in one breast the child continues nursing on the other breast it is important that mother and child are strictly separated except for the brief periods of nursing. The child should be nursed only if the diseased breast is well bandaged. Since the quantities of milk which the child thus receives are frequently no longer adequate because of the severity of the disease and the fact that only one breast is offered, it might be necessary to institute mixed feeding. A woman who had already suffered a severe case of mastitis from a previous child should not be permitted to nurse the next child on the previously diseased breast.

Nursing Difficulties on the Part of the Child

If a child does not develop normally on breast milk and if the amounts of milk taken in do not cover the requirements of the infant the cause, particularly during the first months of life, might lie not only in the mother but also in the child. The cause might erroneously be sought in a deficiency of the breast while actually the sucking infant does not know how to utilize the source of the milk. If after the feeding of an infant which does not show satisfactory development larger quantities of milk can still be expressed from the breast the reason for the lack of development is due essentially to difficulties in nursing on the part of the infant.

The large number of badly nursing infants can be classified into groups: 1) infants who nurse badly from the beginning; this includes the majority of prematurely born infants and the smaller number of full term infants who nurse weakly; 2) infants with undeveloped sucking reflex and 3) children without appetite, that is, children who in the beginning show satisfactory sucking and food intake but who after a while develop displeasure in sucking and finally refuse the breast altogeher.

In the weakly sucking infants and in those where the complicated sucking mechanism is still incomplete or not yet developed the inability to suck is easily understandable. For such children with primary sucking weakness the nursing technique will either have to be changed or the food requirements will have to be covered by temporary addition of expressed milk or cow's milk mixtures. For details concerning these methods the reader is referred to the chapter on "Feeding of the Prematurely Born Infant" (p. 162). In such circumstances two points must be observed: 1) the child must not be hungry because the nursing obstacle can be overcome only with progressing development and weight gain. 2) The functioning of the glands can be maintained and increased only by regularly applying the stimulation of sucking of the infant, even though it is very weak. Regular emptying of the breast should be performed either by manual expression or by emptying the breast with the help of a breast pump. If these requirements are fulfilled the sucking inability and weakness will disappear with the maturation of the child and with stronger sucking.

The difficulties are considerably greater in full term babies whose sucking reflex is incompletely developed or lacking altogether. There are children who are unable to master the complicated sucking process even with correct nursing technique. Through incomplete emptying of the breast the milk secretion soon comes to a stop. The mothers of such infants become restless and nervous if at every feeding the child hardly grasps the breast or lets go again altogether after a few swallows. The sucking ability in those infants is, however, in no way impaired which can be proven by introducing the finger into the mouth of the infant. However, the repetition of the sucking motion, which is necessary for emptying of the breast, is lacking. The satisfaction in the sucking rhythm, as present in the normal child, and the pleasure connected with the food intake are missing. The drinking process becomes a source of desperation for the mother. For the physician it might be easy to persuade the mother to continue putting the child on the breast. But even the most sincere desire for nursing will be destroyed in the mother through the unsuccessful efforts which are repeated at every meal. In some respects the behaviour of these young infants and their indifference towards food intake resembles the behaviour of some older children who eat badly and show no pleasure in eating. It is peculiar however, that these children frequently take their food from a bottle without difficulties. The result is that the mothers who are not satisfied with their efforts at breast feeding soon switch to complete bottle feeding.

In women who are well able to nurse it should be possible to overcome this critical period. In women who from the beginning do not produce milk adequately the prospects are considerably worse. In any case, only calmness and persistence will be of help. The recommendation to extend the periods of nursing over half an hour or even an hour is not sound advice. Sufficient periods of rest between feedings are important for mother and child. Even in these cases the meal should not be extended beyond 10 to 15 minutes. The mother must be made to understand that nothing is lost if nursing does not succeed immediately as she might expect from experience with other children or from reading explanatory texts. The mother must be convinced that the child is not harmed. It is therefore necessary, apart from the nursing attempts, to give the child manually expressed breast milk or to complement the food intake by the addition of cow's milk. At times it is of advantage to forego all nursing attempts for a few days and to feed the child only with manually expressed milk. If the infant receives sufficient nourishment through either of these methods he will show a weight gain which will console the despairing mother and strengthen her self-confidence to continue nursing. If mother and child were already disappointed through unsuccessful nursing attempts they might sometimes be successful after such a breathing spell. The attempts to overcome these difficulties by more frequent feedings seem to be less successful however. In this manner children with undeveloped sucking reflex do not learn to suck on the breast. If the physician decides to add food the quantity should scarcely cover the requirements and should be given in concentrated form e.g., whole milk with 10% sugar mixture.

The third group, the poorly drinking children, usually present no difficulties with sucking and drinking in the beginning. One day, usually during the second or third month, the breast is suddenly refused. The children cease to make swallowing motions and their behaviour and facial expression denote an aversion against the breast; this is in contrast to the children of the second group. The refusal is accompanied by crying and restlessness. It appears very difficult to determine the cause for this behaviour which, one might say, is unnatural. If one does not wish to deviate to the exaggerated explanation of the *Freudian* theories it is perhaps correct to assume that this refusal is based on unpleasant experiences on the part of the child. An awkward position while nursing, causing the child pain, a feeling of suffocation during nursing, a different taste in the milk, a different soap, a different perfume on the mother, might all perhaps bring about unpleasant memories. Such experiences are particularly long retained because the children in such cases are almost always neuropathic, they are easily impressed and their impressions are long-lasting. Here, too, it is occasionally possible to overcome the difficulties if the mother is calm and persistent and one of the enumerated possible causes is recognized and removed. In such cases also a few days' interruption is advisable during which the child is fed with expressed breast milk, possibly supplemented by cow's milk. Before renewing the attempts to put the child on the breast one should in the first days give a light sedative (phenobarbital tablets). Frequently, however, all these attempts are unsuccessful. The refusal of the child makes a resumption of breast feeding impossible or the mother has in the meantime observed that the child will develop satisfactorily even with artificial nourishment without the psychological stress caused by futile nursing attempts.

Among organic obstacles hare lip and cleft palate can make sucking difficult. In these anomalies it must be decided for each individual case whether the ability to nurse exists. It is wrong to assume in advance that such children are unable to take the breast. Children with one-sided and bilateral hare lip are almost always able to accomplish a closure of the oral cavity which is necessary for sucking, by filling the spaces with part of the mother's breast. Even some of the children afflicted with partial or even complete cleft palate are still able to empty the breast adequately.

Congenital heart troubles which already during or soon after birth lead to cyanosis and dyspnea are occasionally a serious obstacle to breast feeding. Injuries or formation of ulcers in the oral cavity (Bednar's aphthae) can temporarily make nursing difficult. By applying dermatol and powdered sugar and possibly also small quantities of anesthetic before meals the wounds will heal and thus the nursing difficulties can be overcome. Teeth which occasionally have erupted at the time of birth do not disturb the sucking. They need not to be removed.

Obstruction of breathing through the nose might become an obstacle in nursing. The most frequent cause is the cold of the infant. During nursing on the breast the child develops a fear of suffocation since breathing through the mouth is impossible during sucking. By inserting two to three drops of adrenalin solution 1:1000 and applying an ointment which does not contain menthol the nasal passage can be freed sufficiently for the short period of nursing so that the meal can be taken, if needed with interruptions. However, a cold in the infant can become such a serious obstacle in regular nursing that the child should be exposed as little as possible to infections by taking preventive measures such as isolation of the child or wearing of a mask if mother or nurse suffers from an infection.

Another serious obstacle in nursing is the not infrequently occurring unilateral or bilateral, complete or almost complete atresia of the choanae. The obstruction of nasal respiration which is caused by this developmental malformation makes it impossible for the infant to accomplish the closing of the lips around the nipple, which is necessary for sucking. The existence of atresia of the choanae is proven by the inability to introduce a thin rubber catheter through the nostrils into the throat.

The criterion for successful feeding of the infant on the breast is never the quantity of milk taken in but only the normal development of the child in the sense of eutrophia (see below, p. 182). Judging nutritional successes on the basis of milk intake can lead to errors. There are children showing satisfactory weight gain on relatively small quantities of breast milk which, however, is very rich in fat, while other children drink relatively large quantities of milk without showing satisfactory development. The simplest criterion for normal development which can be determined objectively is regular control of body weight. If the physiological weight loss of the first days of life is gained back again after two to three weeks the normally developing breast fed infant should gain 150 to 250g or more per week. The constancy in weight gain can, however, be observed only if the child is weighed about once a week. The shorter the intervals between weighings the greater will be the variations in gains in the breast fed infant. This unevenness in gain which is found in too frequent—e.g., daily—weighings might worry the mother who expects an unvarying daily weight gain. The weight gain of the child should therefore be determined only once a week. Daily weighing or weighing after every meal is superfluous. They are permitted only when the actual amount of milk intake is to be determined in a child showing no weight gain in order to obtain a basis for further recommendations, such as addition of other foods etc. Signs of development other than increase in body weight are determination of growth in length, development of adipose tissues, and control of tonus, turgor and color of skin. In the well developing breast fed child the summation of these observations gives us the picture of the healthy, eutrophic infant. The final proof of good health, however, is also in the breast fed infant the ability to overcome unavoidable infections. The first catarrh of the air passages, the first febrile cold must be overcome by the well developing breast fed child within three to four days, without complications.

Parallel to good physical development goes the psychological development. The appearance of psychological achievements at certain ages are as important for judging normal development as the gain in weight. The healthy infant is psychologically well balanced and satisfied. After the child has become adjusted to five meals a day at two to six weeks of age, depending on the child's temperament, he will sleep throughout the greater part of the day and awaken only a short time before the next meal in order to announce his hunger by crying. Or the child is awake and lies quietly in his bed, playing with his fingers and more or less consciously observing his surroundings. Only few children are in the habit of being awake and crying during a certain hour of the day, without any apparent cause for this restlessness which is repeated daily around the same time.

Today almost the only source of breast milk available to the infant is that of his own mother. It is the task and the duty of every physician and every organization supervising the upbringing and nutrition of young children to promote natural feeding by the mother.

> It is no longer timely for the wet nurse to take the place of the mother. In practice the physician will very rarely have the opportunity to give his judgment in the choice of a wet nurse. Only in very few infant homes do we occasionally find women who apart from nursing their own child offer expressed milk for other children. Putting the child on the breast of a wet nurse has now been abandoned even in institutions. Fashion trends, changes in economic conditions and last but not least progressing successes with artificial feeding have brought about the disappearance of the professional wet nurse. If for any reason a child should be fed with breast milk, either in an institution or in the home, the child must not be put on the breast of the donor. This procedure has been abandoned because despite all precautions syphilitic infections of the wet nurse have at times occurred. The wet nurse, after nursing her own child, expresses her milk either manually or with a breast pump; the milk is briefly boiled and then given to other children.

In most large cities there are today centers for collecting breast milk, where the milk of healthy mothers is collected, against payment, to be used for feeding other infants. The treatment of the milk to guarantee the absence of germs (pasteurizing, boiling, etc.) varies in the different collecting stations. The requirements for such breast milk are: a number of germs not exceeding 1000–5000, acidity of about 4° Donics (1 Donic = 0.1g lactic acid per liter) and a specific gravity of 1.030. By boiling the milk the protein bodies are partially denatured, the lipase is destroyed and the taste of the milk is changed. There is, however, no essential difference in nutritional results between breast milk which the infant drinks directly from the breast and breast milk which comes from a collecting station.

The various difficulties which might develop at the beginning of the nursing period or which occasionally appear in later weeks and months should not be taken too seriously or wrongly interpreted by the mother and her surroundings. Among the symptoms which in this respect might be wrongly interpreted through ignorance are first of all the appearance of the stool and a number of symptoms originating with the stomach. Regurgitation and vomiting in the infant are regarded as signs of disorder of the stomach. Constipation is considered as cause for various expressions in the infant denoting discomfort. Diarrhea, on the other hand, is believed a sign of pathological gastro-intestinal processes. All these symptoms require changes in nutrition only if they are accompanied by disturbances in the development of the child. Regurgitation, vomiting, diarrhea and constipation do not permit a diagnosis of nutritional disturbance, in the breast fed child even less than in the bottle fed child. These symptoms should never be the cause for radical changes in the nutritional procedure.

Regurgitation and vomiting in the breast fed infant. Regurgitation and vomiting in the breast fed infant are hardly ever signs of an organic disorder of the stomach. They are, furthermore, only in part the expression of a special irritability of the vomiting center. Light regurgitation of small quantities of milk either immediately following the meal or of a clear fluid interspersed with a few casein lumps even several hours after the feeding are physiological processes. In some children this regurgitation and vomiting occurs from the first days of life. In other infants it develops only after the milk secretion and thus the quantity of the meal has increased. It usually disappears around the third to fourth month of life. The vomiting frequently occurs in children who drink hastily and in large quantities, if the milk secretion is easily flowing and abundant. In other infants the reason is an awkwardness in sucking whereby the infant drinks hastily and swallows much air together with the milk; on eructation the rising air then carries along small amounts of gastric contents. A third group of infants shows no apparent cause for vomiting. In general the physician should not place too much emphasis on the vomiting of breast fed infants so long as the children show a healthy development. Experience has shown that these gastric symptoms usually disappear in the second quarter of the first year so that no therapy is indicated.

The parents should be assured that within a short time, with the introduction of more solid food at the latest, these bothersome symptoms will cease. Only when the quantities of food lost through regurgitation and vomiting are so large that the child shows no gain or possibly even a loss of weight, the vomiting must be treated. Any therapy which attempts to combat the never existing functional or secretional disturbances of the stomach through hydrochloric acid and pepsin or gastric lavage will be unsuccessful. It is just as erroneous to expect to cure the vomiting by relieving the stomach by limiting the intake to tea and cereal water. Weaning from the breast and change in nutrition are of no influence. Even under the assumption that these symptoms appear on a neuropathic basis not much is gained because vomiting breast fed infants show no other characteristic nervous symptoms. It is always necessary to consider the presence of an incipient pylorospasm and to look for other symptoms pointing in that direction since habitual vomiting of the breast fed child during the first weeks of life occasionally precedes real pylorospasm. If the vomiting persists other nervous or organic causes must also be considered (esophagospasm, hiatus hernia, etc. see later p. 207).

In the treatment of harmless vomiting it is occasionally of advantage to stimulate the so-called peristolic function of the stomach, that is, "the ability of the stomach to contract around its contents." Occasionally this reflex is not sufficiently developed in the infant and the stimulation of liquid milk does not suffice to bring about the peristole of the gastric walls. Only when solid food is added to the diet is it possible to awaken the peristolic function of the stomach. By offering small quantities of somewhat thicker pureed food before the regular liquid diet vomiting will cease in a number of patients. The cereal can be made from expressed breast milk and farina. It is also perfectly permissible to offer even infants during the first weeks of life small quantities of cereal prepared with cow's milk (100g whole milk, one to one and a half teaspoons farina, one to one and a half teaspoons sugar, boiled for 20 minutes), in amounts of three to four teaspoons before every meal. Aside from this it is advisable to depress the irritability of the nervous centers with narcotics and sedatives (e.g., luminal 0.015 t.i.d. or similar). Narcotica are indicated particularly in restless, frequently crying infants. The irritability of nerve tracts of the vagus is diminished through medication of atropine, or better perhaps Eumydrin (both in a solution of 1/1000, two to four drops daily) shortly before the meal. Mayerhofer recommends Papaverin hydrochloride 0.01, Pyramidon 0.05 three times daily before the meal. It is usually possible to gradually improve even more severe vomiting by offering the thick cereal before the milk and by regular administration of one of the above medications. It is necessary to continue this treatment for a longer period of time (Fig. 11).

Singultus appears frequently in the young infant; it occurs following the meal and can continue for a long time. The cause is probably a disturbance of the nerves of the diaphragm due to hasty drinking or a raising of the diaphragm if the stomach becomes excessively filled with food and air. In order to terminate this condition, which strangely enough hardly disturbs the infant, it sometimes suffices to place the child upright so that the air can escape from the stomach. In other children a little sugar placed on the tongue, or causing the child to cry, will bring relief.

Constipation or, more correctly, rare stool elimination (because frequently even after a pause of several days the stool is not hard but soft and pasty) is relatively rare in the breast fed infant during the first days of life. Infrequent bowel movements usually occur only in the second month of life. During the first weeks mild diarrheas are more frequently observed. At the age of four to six weeks the stools become rarer. This change might cause a new neurosis in the mother. Since a considerable number of nursing mothers suffer from real constipation they frequently begin to wonder whether this might not be the beginning of a hereditary and annoying disturbance in the child. As a result the mother, in reporting in the history of the child about

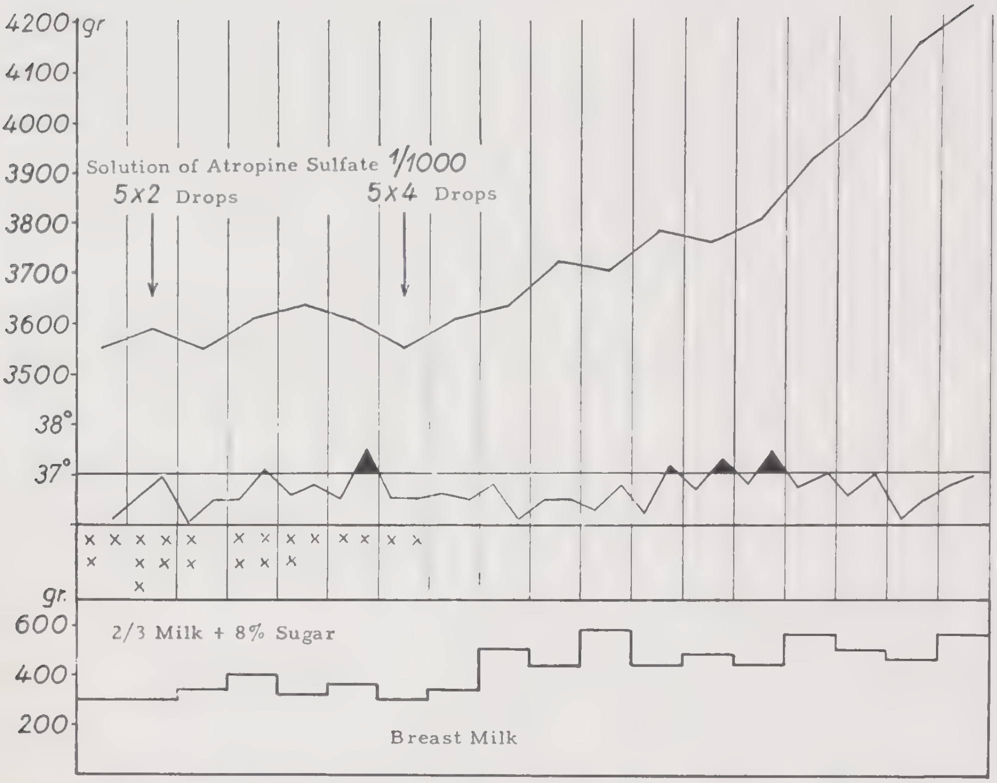


Fig. 11. Infant P.M., two months old. Birth weight 3950 g. Diet: Breast milk plus 2/3 milk plus 8% sugar. Vomiting after each meal for one month. Habitual vomiting (no visible peristalsis of the stomach, etc.). The roentgenogram shows retarded emptying of the stomach. Treatment with atropine sulfate solution.

the "bad stools of her infant" does not refer, as the physician believes, to diarrheal stools but rather to infrequent bowel movements. Under "good stool" the mother understands the daily production of two to three pasty stools of more or less thin consistency. The pseudo-constipation of the breast fed infant can become quite extensive. Up to a week can pass before the children produce a stool which even after such a long time is not very large in quantity. In spite of this the behaviour and development of the children is completely undisturbed. This finding occurs much more rarely in an institution than in a private home. In the private home the physician is frequently forced to do something against this "pseudo-constipation" since most adults, judging from the importance and careful observation of their own, if possible, daily bowel movements, consider the absence of a daily stool in the infant a serious and pathological symptom.

The reason for these rare stools is the almost complete absorption of breast milk in the intestinal tract of the child, without leaving hardly any waste. This complete absorption of breast milk in the small intestines is difficult to explain. Frequently the incompletely developed ability to expel the stools from the lowest sections of the intestines leads to pseudo-obstipation. Young infants have not yet learned to coordinate the large quantity of smooth and striated muscles which are necessary for expelling the stool. The apparent strain of the infant during a bowel movement whereby the face of the child becomes intensively red even though the stools are entirely soft, demonstrates the awkwardness with which this process is undertaken.

Before diagnosing a harmless constipation it is important to first consider the serious diseases which can cause constipation at this age. Tumors, malformations, partial atresia of the anus, Hirschsprung's disease, myxedema and other conditions must be considered (see p. 468). If the rare occurrence of stools turns out to be harmless it will be necessary in the private home to institute some treatment since the condition worries the mother considerably. It is, in any case, preferable if the physician gives the orders than if the nervous mother takes up the fight against this "constipation" on her own. Enemas of any kind are not advisable in the treatment of constipation of the breast fed infant. The medication of cathartics (such as castor oil etc.) can turn this harmless condition into a serious diarrhea. The purpose of the treatment is to increase the waste products in the intestines. This increases the quantities of the stools and this in turn increases the stimulation on the walls of the large intestines. It can usually be accomplished by adding two to four teaspoons malt extract or apple sauce or raw fruit juice. Similar results can be obtained by the early addition of cereals or pureed fruits. As the quantity of additional food increases in the course of the months, the "constipation" becomes rarer. If constipation or retention of stools occurs in later months of life one must consider fissures or rhagades in the anus or hemorrhoids which occur not infrequently already during infancy, as cause for this holding back of the stools.

Diarrhea. The expulsion of stools in increased quantities, of pasty liquid to liquid consistency, green and mucous, occurs not infrequently in the healthy breast fed infant. It occurs at least as frequently as the discharge of paste-like, golden yellow stools which are always described as the "normal stools of the breast fed infant." The appearance of loose stools may lead to superfluous and harmful therapy. In the breast fed infant diarrheas appear so frequently during the first period of life, in institutions as well as in private homes that hardly any child is spared. These so-called initial diarrheas begin already during the middle or end of the first or during the second week of life. They continue for about four, six or eight weeks and are followed by rarer stools. The general condition of the child is not affected by the presence of these diarrheas. The weight gain is satisfactory and the appearance of the children is fresh and rosy. Only the skin on the buttocks is irritated by the frequent liquid stools and intertrigo may result. This irritation heals rapidly by treating it with protective salves (zinc ointment, zinc oil, Desitin and powder) and by using oil (paraffin oil) in place of water for cleaning the buttocks, and at times also by applying a 5% gentian violet solution or, in deeper ulceration, balsam of Peru. The skin irritation is the result of maceration.

Frequent discharges of stools are encountered even after the newborn period more frequently in healthy infants who are raised in institutions. In explaining the tendency to diarrhea in institutions the customary nursing procedures, such as taking rectal temperatures daily, are held responsible. But even if one foregoes taking the temperatures the incidence of diarrhea hardly decreases.

In newborn and infant departments, under ideal nursing conditions and with well trained and adequate nursing personnel, this disadvantage of nursing on a large scale decreases and disappears altogether. This is shown by the results of aseptic care of prematures in closed incubators (see p. 165). Several factors indicate that in the last analysis diarrheas in young children are caused by the difficulties in producing a physiological bacterial flora in the intestinal tract. The numerous and varied bacteria which the young infant, raised in large institutions, must absorb and tolerate in the gastro-intestinal tract cause an irritation of the intestinal wall which expresses itself in the so-called *irritative diarrhea* (*Finkelstein*). For successful artificial feeding of young children especially in institutions, it is essential to recognize the harmlessness of these diarrheas. As long as this type of diarrhea was again and again subjected to intensive treatment, as long as hunger and starvation cures were ordered, a success in artificial feeding of young children was impossible. The unfavorable results which in former years were observed every place where young infants were raised in institutions can to a great extent be traced back to a wrong evaluation of diarrhea and the ensuing hunger therapy. Only by observing the behaviour of the infant, the presence or absence of signs of damage to the general condition, can we obtain information for the evaluation and treatment of these conditions.

These diarrheas originate as a result of a state of irritation in the lowest sections of the large intestines. The irritation of the intestinal wall causes a more rapid peristalsis of the large intestines. This prevents the normal thickening of the stool which is thus discharged in more or less liquid form. Too frequent taking of the temperature in the rectum can also cause such intestinal irritations (see p. 136).

Treatment of this initial diarrhea of the breast fed infant is superfluous. After a few weeks the diarrhea ceases spontaneously. In the care of a closed institution it is possible to wait without treatment. But also in the private home the parents should be assured that these loose stools are harmless and temporary so long as the development of the child is not affected. The physician must prevent weaning or radical changes in nutrition on account of these diarrheas. Mothers who believe that the loose stools are due to an inferior quality of their milk have a tendency towards such procedures. But also the physician who explains the diarrhea as a result of overfeeding and limits the amount of milk intake acts incorrectly. In case of diarrhea in the breast fed infant a change in nutrition is necessary only if simultaneously the child stops gaining weight and shows other pathological symptoms. In that case, however, it is no longer a harmless initial diarrhea of the breast fed infant but a nutritional disturbance which must be treated according to the methods applying for such diseases (see p. 240). The lack of action in beginning diarrheas on the part of the physician will frequently be opposed by the mother who might demand active treatment. In such cases corrections should be made by the simplest possible means, certainly without any radical changes in nutrition or nursing technique. Addition of small quantities of protein milk with sugar, one-half to one teaspoon of some protein preparation (e.g., Casec), dissolved in alkali salt solution, suffice to correct the appearance of the stool. Furthermore small quantities of absorbent or adstringent remedies, e.g., three to four times daily a fourth teaspoon dermatol, Bolus alba 2%, carob flour (Arabon, Ceraton etc.) in small quantities of expressed milk with each meal will improve the number and appearance of the stools.

MIXED FEEDING. WEANING

If the quantities of breast milk are not sufficient to cover the requirements of the infant the food must be supplemented by the addition of some artificial food. The addition of food for covering the requirements becomes necessary if the healthy infant does not at all or not sufficiently gain in weight over a period of two or three weeks at the most. As food supplement a mixture is chosen which corresponds to the age of the child. The addition of food does not follow a routine but if a control of the weight of every meal shows that only about 20–50g are lacking to cover the requirements, those small quantities are not given from the bottle but with a spoon after nursing. If the deficit is larger it is usually more advisable to have the infant nurse from both breasts for only two or three of the daily meals and give him two or three entire meals of artificial food which is, according to the child's age, offered either through the bottle or, if more solid food is given, with the spoon or from the cup. The beginning of additional food intake marks for a number of infants the end of breast feeding. The children get used to drinking from the bottle which is less strenuous.

In the interest of mother and child nursing should not be continued beyond the sixth or seventh month of life. After this time the quality of natural nutrition no longer covers the requirements of the child. The results might be a stoppage in weight gain, hypoproteinemia or anemia. Weaning of the infant should never take place suddenly unless compelling reasons on the part of mother or child necessitate a sudden change from natural to artificial feeding. If time and circumstances permit, the child should be weaned in such a manner that about every third to seventh day one breast milk meal after the other is replaced by artificial food. In every case an entire meal should be replaced immediately by the new diet. Opposition on the part of the child is thus easily overcome. After about two to three weeks the child has changed over completely to a diet which corresponds to his age. If the breasts continue to secrete milk abundantly the mother should drink very little during the weaning period in order to decrease the quantity of milk. A light cathartic might also bring about a decrease in milk supply in the mother. A more rapid cessation of the milk flow can be accomplished by giving Stilbestrol. The breasts should be tied up during the period of weaning. Such measures for terminating the milk secretion are important for those women who wean their children with regret and who shows a tendency to resume breat feeding as long as the breast glands continue secreting and who consider the preparation of artificial milk mixtures as troublesome.

ARTIFICIAL FEEDING OF THE HEALTHY INFANT

Principles of Artificial Feeding

As little as forty years ago this chapter would have started with the discouraging sentence that any artificial feeding of the infant was a dangerous undertaking which would be successful in only a few cases. The mortality rate of bottle fed infants, which was then seven times higher than that of breast fed infants, justified this scepticism. Today it is understood that good nutritional results can be obtained in most bottle fed infants under somewhat organized and adequate hygienic external conditions. The assumption that artificial nutrition in itself has a harmful effect on the child must, therefore, be abandoned. Today artificial feeding may no longer be presented as convenient excuse by the physician to explain lack of nutritional success. However, it should not diminish the important propaganda for breast feeding. *Breast feeding is and remains the best feeding method as intended by nature for raising young infants.* It should be given up only if natural feeding cannot be carried out for some special reasons, if it is hopeless or not indicated. This requirement becomes the more imperative the lower the social conditions are under which the infant is raised. All statistics reveal the same conclusion, that the smaller the family income, the smaller and unfavorable the apartment, the lower the rent, the more primitive the occupation of the father, the higher will be the mortality rate with artificial feeding as compared to breast feeding. Illegitimacy of the child with its social implications, lowers even today the chances of survival with artificial nutrition. Under socially and economically adequate conditions, with some understanding on the part of the parents for hygienic regulations and with the existence of means and possibilities to carry out the orders of the physician, it is not difficult to obtain satisfactory development with an artificial diet.

The conditions for obtaining good results with artificial feeding of an infant are: continuous and regular supervision and preventive care which is not necessary to the same extent in breast fed children. Since the composition and quantity of the food are no longer within the framework of a natural process and are left to human discretion mistakes can easily occur. In choosing and measuring the artificial food it must be attempted not only to obtain normal functioning of the gastro-intestinal tract but the development of the organism must be such that it will be as resistant to any strain and infection as the organism of the breast fed infant. The success of artificial feeding cannot be measured only by a satisfactory weight gain of the child. Only then do the results of artificial feeding correspond to those of breast feeding when vitality and resistance are as high as with natural nutrition, especially up to the end of the first year of life.

For many years attempts have been made to explain the differences in nutritional successes with cow's milk and breast milk through a difference in the chemical composition of the two types of milk. At various times one or the other of the components of milk were held responsible for this (see Chapter on Milk). While there are no doubt physico-chemical and biological differences, particularly regarding the composition of whey and of protein, this branch of research has not decisively influenced the practice of artificial feeding despite all theoretical results. The conclusions obtained in this one-sided attitude of research work have even at times presented some danger to the infants. For the exclusion or avoidance of a substance which at one point was particularly feared, or rather the one-sided increase of substances which at the moment were considered harmless resulted frequently in an unbalanced diet which in its composition no longer complied with the requirements of the organism. Many apparent difficulties and many a damage which resulted from this one-sided attitude to the practice of nutrition could have been avoided, as can now be stated in retrospect.

The following examples of formerly recommended methods of artificial nutrition will justify the above statement: the recommendation to offer food mixtures containing little or no fat was bound to be just as unsuccessful in the healthy child as mixtures with strongly reduced whey or carbohydrate content. Despite fat deficiency in the food it is possible to obtain satisfactory weight gains provided this deficiency is compensated by abundant carbohydrate intake. In the long run, however, the solid building up of the organism, immunity and development of adipose tissues must be affected by the deficiency of the special nutritional value of fat. Reduction in whey content was frequently connected with excessive dilution of the food and thus in practice brought about dangers of undernutrition. Excessive caution in carbohydrate administration, particularly sugar, resulted in the lack of a substance which is essential for heat regulation and for growth. The carbohydrate, as was already mentioned previously, is an irreplaceable substance for fixing fluids in the cells and thus for promoting growth. Case histories of previous years, judged from the modern point of view, show that a number of nutritional failures which were explained with intolerance of cow's milk or constitutional inability of the child to digest food, can now be properly explained by the faulty composition of the food based on one-sided and incorrect assumptions.

This applies similarly to experiments which have recently been undertaken to obtain through special preparation of the artificial food a bacterial flora in the intestinal tract (bac. bifidus) which is characteristic for the breast fed infant. A changing of the bacterial flora will not decisively influence the results of artificial nutrition.

The differences between natural and artificial feeding are:

1) Cow's milk, as compared to breast milk, remains longer in the stomach and also requires more time for the total passage of food through the intestinal tract.
2) Cow's milk requires a more active secretion on the part of the intestinal tract and glands such as liver, pancreas, etc.
3) Cow's milk lacks lipase which is contained in breast milk and which promotes digestion of fat.

All these as well as some other minor differences do not suffice, however, to explain in the long run the poorer nutritional results with cow's milk feeding.

Only after abandoning all attempts to find the cause for the lack of success with artificial feeding in any single substance in the cow's milk important practical progress was possible in artificial feeding. Today the essential principles of nutrition are: 1) not a single substance contained in the natural prototype of any infant nutrition—breast milk—may be withheld from the child for any length of time; 2) the quantitative relationship of the components should approximate that of breast milk. All mixtures containing protein, fat and carbohydrates in approximately the same relationship as breast milk can, therefore, be used for feeding the healthy infant over longer periods of time; 3) adequate intake of water, salts and vitamins is a further requirement for effective artificial nutrition. Any food mixture deviating essentially from the above principles is not suitable for long-term feeding of infants. It is understood that in the sick child changes from this pattern can and must be made (see later, Nutritional therapy). But here, too, an earliest possible return to a complete long-term feeding schedule is indicated. While in the older child and in the adult damages caused by one-sided nutrition appear only after a longer period of time, the infant shows disturbances caused by a deficiency of one substance or another already after a few weeks. The success of artificial nutrition should therefore not be expected on the basis of one single method. Satisfactory nutritional results can be obtained by seemingly quite different methods provided the quantitative and qualitative requirements of the organism are always fully met.

Artificial food must contain all the substances found in breast milk. The relationship of the most important substances should approximate that of breast milk. The proportion of protein, fat and carbohydrate is in breast milk 1:3.5:7. The relationship of fat to carbohydrate (1:2) is practically the most important since there are a number of reciprocal connections between fat and carbohydrate metabolism. Fat and carbohydrate are antagonists in the organic metabolism. The slowly burning fat reduces metabolism, while the rapidly burning carbohydrate increases this process. Fat impedes water storage, carbohydrates promote it. Fat causes stable, carbohydrates labile tissue structure. Fat and the lipoids and vitamins bound to the fat increase immunity and resistance while excessive intake of carbohydrate diminishes them through increased water storage. It is one of the tasks of an adequate artificial diet to bring about a balance of these antagonisms. Empirically this occurs when for every part of fat two parts of carbohydrate are present. The fat requirements of the infant amount to 3 to 4g per kilogram of body weight, the carbohydrate requirements to 8 to 10g per kilogram of body weight.

The amount of protein content in the food affects the interplay of nutritional substances to a lesser degree. Even if the milk is strongly diluted the protein requirements are still met. It has not yet been determined whether an intake of protein greatly exceeding the requirements is harmful or not. Damages caused by excessive protein intake have not been proven. Symptoms of supposed damage due to excess protein (pallor, meteorism, hypotonia etc.) can be traced back to a lack of other substances (carbohydrates) rather than to excessive protein intake. In any case the artificial food of the healthy infant should not unnecessarily contain excessive amounts of protein. About 2 or 3g at the most per kilogram of body weight are adequate. For therapeutic reasons a high protein diet might occasionally be permitted or even indicated.

By making cow's milk whey similar to that of breast milk it has also been attempted to avoid the disadvantages of artificial nutrition. The composition of whey, its varying ion content and perhaps also its varying albumin content is undoubtedly of importance for the physico-chemical processes in and beyond the intestinal tract and for water fixation.

> Even if one does not wish to accept as proof the results of the experiments in the exchange of whey (disturbances after intake of cow's milk whey plus breast milk casein plus breast milk fat, which did not occur if changed to breast milk whey plus cow's milk fat plus a cow's milk casein), the differences between type-specific and hetero-specific whey with regard to nutritional successes have been proven. The whey, and even the part of it which is free of albumin, has a considerable influence upon the speed of absorption of food from stomach and intestines. It is probable that also the bacterial flora in the intestinal tract and a number of fermentative processes (see above) are influenced by the composition of whey.

In the healthy child cow's milk whey is not expected to exert a harmful influence on the nutritional results. The amounts of salts required for growth are met by the whey even in a milk dilution of 1:2. But even a considerably higher mineral intake which exceeds by far the mineral content of breast milk is harmless Attempts to adapt cow's milk whey to breast milk whey are unnecessary in the nutrition of the healthy child.

Vitamin A is present in adequate quantities in cow's milk as well as in breast milk provided the dilution of cow's milk does not exceed 1:2. The amounts of the vitamin B complex contained in cow's milk are sufficient during the first weeks. Later they must be supplemented by foods rich in vitamin B. The quantities of scurvy-preventing vitamin C contained in the milk of adequately fed animals are sufficient for the child, provided the milk has been handled correctly. Such cow's milk contains about 5–15 mg vitamin C per 100 cc. These quantities are sufficient to meet the daily vitamin C requirements of the infant (about 50 mg). Only after the fourth month of life the question of insufficient vitamin C intake becomes of practical significance. The increased vitamin C requirements will then be met by addition of vitamin C containing food (fresh fruit juices, vegetables). Neither breast milk nor cow's milk contains vitamin D in sufficient quantities to meet the requirements of the infant after the third month of life. This deficit should be compensated by prophylactic measures (see chap. on Rickets).

In one respect the customary artificial food mixtures deviate considerably from the model of the breast milk. Experience has shown for a long time that carbohydrates in cow's milk mixtures are better tolerated if they are not given in form of crystalline sugar. While the intake of flour during the first weeks of life was formerly rejected because of assumed indigestibility we today use already beginning with the second week of life strained flour water and later flour itself cooked in water for diluting cow's milk. In the first weeks of life the flour digesting ferment is absent in the intestines so that in case of flour intake the stools of young infants show an abundance of material staining with iodine. However, the ferment appears within a few days already following the repeated stimulation of flour intake. The quantity of iodine stained material decreases then from day to day and gradually disappears altogether. Since, however, the possibility must always be considered that undigested flour passes through the small intestines into the lower sections of the intestinal tract it is customary not to offer flour to the youngest infants, up to about two months of age. Strained cereal water or roasted flour, however, can be given already from the second week of life without danger.

> Practical experience has preceded the theoretical evaluation of the efficacy of this so-called second carbohydrate. The substitution of even part of the crystalline sugar by this second carbohydrate frequently causes increased weight gain of the child even though it is not connected with an increase in calories of the food. Not only are flour and sugar of unequal value with regard to the metabolic processes, but even the different types of flour show variations among each other. The amount of the cathartic effect should depend on the degree of bacterial decomposition of the flour in the intestinal tract which is greater in oat-flour than in wheat-flour, and in wheat-flour greater than in rice-flour.

The roasting processes too seem to be of significance for better tolerance of the different types of flour. Through the roasting process, e.g., roasted flour, substances are developed in the flour which are noticeable by their agreeable smell. These aromatic substances seem to stimulate metabolism. According to all practical experience roasted flour is undoubtedly superior. After all, the frequently recommended and used infant flours are nothing but flours which are changed through a roasting process and are similar to roasted and ground zwieback.

ABSORPTION AND FOOD METABOLISM

Theoretically the most recommendable artificial food mixtures are those which are as rapidly absorbed by the intestinal tract as breast milk. The best protection against the development of pathological processes in the digestive system is rapid absorption from and regular emptying of the small intestines. The process of food absorption is complicated by the fact that the absorption of each individual substance does not follow its own laws but that the substances contained in a mixture influence each other considerably.

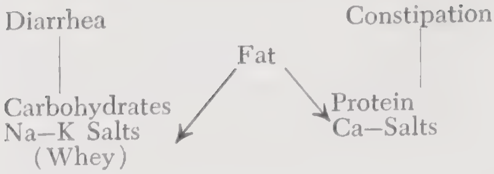
To give the example of one single substance, sugar can for instance very rapidly be proven in the blood stream. Orally taken sugar reaches the blood already within 20 minutes and its absorption is completed after about 45 minutes. As soon as another substance is added to the sugar the curve of the blood sugar changes considerably. There is a delay, which is greater in case of addition of hetero-specific whey or fat than of protein. The speed of absorption of a food mixture from the intestinal tract therefore depends more on the mixture, the so-called correlation of nutritional substances, than on the absolute quantities of the substances contained in the mixture.

Because of rapid absorption strong dilutions are preferable to whole milk rich in whey and fat. For practical nutrition, however, the disadvantage of less diluted milk with regard to absorption cannot be taken into account since it is difficult to meet the nutritional requirements with strongly diluted and thus low-caloric mixtures, particularly in the youngest children.

The appearance of the stool depends essentially on the choice and dosage of carbohydrates and the quantities of protein. The carbohydrates themselves are quite dissimilar with regard to speed or absorption. There are rapidly and slowly absorbable carbohydrates. The following sequence can be set up with regard to speed of absorption: rice-flour is slower absorbed than wheat flour, wheat flour slower than oat flour, oat flour slower than milk sugar, milk sugar slower than malt extract, malt extract slower than beet sugar, and beet sugar slower than dextrin maltose. In the final analysis the appearance of the stool depends on the protein and carbohydrate intake. It is therefore easy to obtain in the healthy child more liquid or solid stools by modifying the food intake in one direction or the other.

Apart from protein and carbohydrates the fat and whey components also participate in the modification of the stool. If the stools are normal and solid the fat content of the food can be increased without fear of diarrhea. If there is a tendency or actual existence of diarrhea, any addition of fat will aggravate the diarrhea through the formation of fatty acids. Whey can also contribute to the development of diarrhea. However, the different salts of the whey again react differently. Sodium and potassium speed up the peristalsis, calcium slows it down. This proves the old practical experience of the constipating effect of calcium salts.

Thus the practice of artificial nutrition forms a pattern which can best be compared to the picture of a scale which symbolizes, when in balance, the "normal" intestinal processes, that is, a condition which is neither constipation nor diarrhea. By upsetting the balance through certain nutritional substances to one side or the other the results are either frequent and loose stools (diarrhea) or rare and solid stools (constipation). Substances promoting diarrhea are, as mentioned above, carbohydrates and the part of whey containing sodium and potassium salts. Protein and calcium salts have a constipating effect. The action of the fats depends on the tendencies prevailing at the moment. At times they promote diarrhea, at other times constipation. The schematic presentation of these processes also applies to therapeutic endeavors to treat diarrhea or constipation, respectively. In the following diagram we thus find the simple picture containing the principles of artificial feeding:



With the complexity of food mixtures matters are never as simple as presented above. A tendency in one direction may be counteracted in the intestinal tract by a tendency in another direction. If a mixture contains large quantities of protein more carbohydrates are tolerated than in mixtures which are poor in protein. With a diet containing large amounts of carbohydrates an increase in fat intake might possibly result in a pathological reaction in the direction of diarrhea, etc.

It is not easy to determine which processes are of advantage to the artificially fed infant. In comparison to the pattern of the breast fed infant it would be correct to assume that the prevalence of moderate fermentation in the intestinal tract would also be indicated for the artificially fed child. However, the transition to a pathological condition occurs much easier, more rapidly and more frequently in the artificially fed child. It is advisable to choose the artificial food mixtures in such a manner that the tendency to diarrhea, fermentation and acid formation is not too strong. On the other hand, a tendency to constipation is hardly of disadvantage, provided that it develops on a correctly balanced diet. The dangers of prevalent putrefaction become apparent only when the composition of the feeding mixture is also unfavorable for growth.

Normally the observation of the character of the stools, whether more alkaline or more acid, whether solid or liquid, is not of as great significance as was previously ascribed to it. It is not the appearance of the stools which is important but the development of the child on a diet with an optimal composition.

Practice of Artificial Feeding

The practical orders of the physician for the artificial nutrition of a healthy child are altogether simple, and the simpler artificial feeding is carried out the better it is. The numerous methods which are usually recommended for artificial nutrition can be summarized in a few simple rules:

1) The amount of food must meet the nutritional requirements of the child (Quantitative sufficiency of food).

2) The composition of the food should be based on the correlation of food substances as contained in breast milk (Correct correlation of nutritional substances).

3) The milk should be supplemented in due time by the addition of suitable foods (Qualitative sufficiency of food).

Food Quantity and Caloric Value

In calculating the nutritional requirements of the child the caloric value of the food must be taken into account. The old rules ($^{1}/_{10}$ of the body weight in milk, $^{1}/_{100}$ of the body weight in sugar or $^{1}/_{6}$ of the body weight in one of the customary feeding mixtures) also give approximate useful indications for calculating the daily quantities of nutrition. These calculations do not apply to the occasionally used concentrated feeding mixtures (whole milk plus 17% sugar, butter and flour mixtures, condensed milk, etc.). Calculations of food requirements are never mathematically exact values. They do not take into account the individual variations of food requirements of infants. They only give an approximate figure for determining the necessary food quantities.

The first task for regulating the artificial feeding of an infant is to supply a quantity sufficient to meet the requirements. Many growth disturbances can be avoided if this first requirement is fulfilled. Experiences of only a few decades ago show that not infrequently, out of fear of artificial food, feeding mixtures were given for weeks which covered only half of the requirements. This, as we understand today, unavoidably resulted in serious disturbances. Unfortunately such occurrences and basic faults in the nutrition of young infants are even today no rarity. By meeting the quantitative requirements one of the most important causes leading to dystrophy or atrophy is eliminated.

In determining the nutrition of the individual infant the correct quantity of food is that on which the child shows adequate, normal weight gain. In this sense the minimal quantity is also the optimal quantity. In any case, one of the basic principles of artificial feeding is not to increase the food quantity as long as weight gain and development correspond to the age of the child. Neither theoretical calculations nor apparent signs of hunger on the part of the child justify a deviation from this rule.

The recommended food quantity is adequate if the healthy child during the first weeks of life following the newborn period attains a gain of at least 150 to 200g per week. Lower average weight gains point toward an insufficient food intake, provided the stools are normal and there are no pathological disturbances. Excessive weight gains indicate over-feeding of the child.

In cases of artificial feeding the system of five meals a day is today an accepted principle during the first four to five months of life, recognized even by those favoring a self-regulation of feeding. The distribution of the meals is such that beginning with 6 or 7 o'clock in the morning until 10 or 11 o'clock at night, food is given regularly every four hours. This is followed by an eight hour rest during the night. The healthy infant rapidly becomes accustomed to this program. According to his temperament he will, after a few days of life or perhaps only after four to six weeks, occupy the time between meals by resting or sleeping. With great regularity he will announce the time for his meals.

Composition of the Food

Any artificial nutrition has for decades been based on the dilution of cow's milk. Even today it is still questionable to which degree this dilution should be carried. The original governing idea was the desire to reduce protein intake to the amount of protein contained in breast milk. Although today the fear of feeding the protein of cow's milk is no longer valid the principle of milk dilution is still advocated in order to avoid unnecessarily high consumption of protein. If whole milk with added sugar is given there is a danger of overfeeding or if the total amount of food intake is small, because of its high caloric value, there is a danger of insufficient water intake. It is of no practical significance whether feeding is begun with $^1/_3$ or $^1/_2$ milk during the first days of life. It seems preferable to start with $^1/_2$ milk.

In order to compensate for the loss of nutritional value due to the dilution of the milk, to re-establish the correct relationship of carbohydrate to fat = 2:1 and to meet the high carbohydrate requirements of the infant it is necessary to supplement the ½ milk dilution with additional foodstuffs.

Addition of carbohydrates. The demands of growth and heat production upon the food require the addition of carbohydrates to the diluted milk. The quantities of additional carbohydrates in turn depend on the relationship of carbohydrates to fat as existing in breast milk (2:1). This requirement also applies to undiluted cow's milk which should not be given without addition of carbohydrates. The formerly so frequent failures on a diet of undiluted cow's milk which were blamed on the nature of heterospecific food, could be traced back solely to the low content of sugar in cow's milk in relationship to the fat content (1:1). Whole milk is a suitable food even for young infants, provided carbohydrates are added. In the healthy infant carbohydrates are added in form of crystalline sugar (cooking sugar). The use of special sugar mixtures is superfluous for healthy children with normal stools. Sugar mixtures have no special nutritional value. Only if there is a tendency to frequent stools special sugar mixtures or dextrin maltose are indicated.

The quantities of carbohydrates to be added amount to 5% for the customary dilutions (½ or ⅔ milk), based on the total amount of food. A deficit of even 1 or 2% below the norm will in the long run usually have harmful effects even if an attempt is made to compensate for the deficit in calorically equal amounts of fat. An increase of the quantity from 5% to 8% or 10% is occasionally indicated if more rapid weight gain is desired or if in cases of poorly drinking infants the food is to be enriched (made more concentrated).

V. Pirquet even recommended an addition of 17% sugar to whole milk. Although such a strong sugar concentration cannot generally be recommended in practice because the composition of the food becomes too rich in carbohydrates (fat: carbohydrates = 1:5) this food which contains twice as many calories as the customary milk mixtures ("double food") can be used for short periods of time for poorly drinking children. The successful experiences with these highly concentrated sugar mixtures have at least had the favorable effect of dispelling the fear that sugar promotes diarrhea in the healthy child.

The *liquid used for diluting* the milk is, after the 12th to 14th day of life, today no longer pure water. Because of the favorable effect of the second carbohydrate the milk is diluted with strained cereal water or flour cooked in water. The choice of flours or cereals for the preparation of the diluting liquid depends on the consistency of the stools. For practical purposes strained oatmeal, corn flour or farina water are recommended if there is a tendency to constipation, or rice water (prepared from whole rice, not from rice flour) in case of tendency to frequent stools. The second carbohydrate is added in quantities of 2 to 3% of the entire food intake. Undoubtedly the water strained off the so-called infant cereals can also be used for the dilution of milk. This is favored by the mothers, not only because of the impressive advertisements but also because these mixtures are easier to prepare than flour water or strained cereal water. Infant cereals require only a few minutes' boiling while for instance rice water needs several hours of preparation.

Most infant cereals contain roasted products of wheat flour which are believed to possess special qualities in the building of cells (special nutritional value). Whatever can be accomplished by the feeding of special infant cereals can also be accomplished by the addition of flour water or flour, beginning with the third to fourth week of life. The special infant cereals themselves vary, like natural flours, in their effect on the peristalsis. Those which are strongly dextrinized have a similar effect as rice flour, farina, or corn flour, that is, rather constipating; the more saccharized infant cereals react more like oatmeal, which is slightly cathartic. As substitute of infant cereal one can use boiled ground zwieback or biscuit flour.

Addition of fat. Satisfactory nutritional results can be obtained with the simple milk-flour-sugar mixtures. However, the customary dilutions have a decreased fat content compared to breast milk. The loss of this substance is compensated, with regard to caloric value, by carbohydrates. By this method the special nutritional value of fat is, however, not replaced in the same value. During the first four weeks of life fat should not be added because of the dangers of diarrhea. After the third or fourth week of life the missing fat should be replaced by addition of 1% butter or margarine, particularly if the fat content of the milk is low (2.5–3% of whole milk used). If the whole milk or canned milk which is used for preparing the infant's food contains 4% fat or more the addition of fat is superfluous.

As basic food for the healthy infant we thus get a simple mixture which in the great majority of infants gives a satisfactory nutritional result with great regularity up to the third month of life. For the first three months of life it consists of:

$^1/_2$ milk, diluted with 2 to 3% cereal water and 5% sugar and 1% fat.

A feeding mixture which complies with all the above mentioned requirements is *Czerny-Kleinschmidt's* butter-flour-food. This is $^1/_2$ or $^2/_5$ milk, enriched with fat, sugar and flour, with fat and flour being added in roasted form. The butter is made more digestible by the removal of volatile fatty acids through intensive heating. Through the roasting process the flour assumes new biochemical qualities which however cannot as yet be clearly defined. An added advantage is that this butter-flour food has a pleasant aroma and taste and is therefore liked by most infants. The butter-flour food is a complete food for the young infant.

Milk Supplements

Beginning with the third month of life the addition of other foods becomes necessary since milk, flour water and sugar alone no longer meet the requirements of the growing organism. As first additional food the infant receives, after the third month, raw fruit juices (orange, lemon, tomato juice, etc.) in quantities of four, later six to ten teaspoons (30 to 50g) distributed over two to three meals. The fear that raw fruit juices can cause diarrhea is unjustified provided that the meaty part of the fruit which contains cellulose is not given. For the practice of nutrition all fruit juices are of equal value. It is never necessary to give two or more different fruit juices. Canned fruit juices, prepared under technically approved methods can also be substituted in the same amounts at times when fresh juices are not available. The fruit juices are best given to the infant in form of a fruit purée which is prepared without milk, from mashed bananas, passed through a sieve, fruit juices and sugar. The quantities of fruit purée which are given lightly warmed correspond to the amount of the milk meal which they replace. If bananas are not available the basis of the fruit purée can be mashed carrots, rapidly cooked in the pressure cooker, boiled rice or farina, with perhaps the addition of biscuit or dry ground zwieback. The addition of white cheese (cream cheese) or scraped apples which spoil the taste of the purée is superfluous.

Around the third or fourth month of life a second bottle of milk, the best is perhaps the noon meal of the day, is replaced by a cereal consisting of 150 to 200g whole milk (undiluted), one to one and one-half teaspoons finely ground farina (special infant food) and one and one-half to two teaspoons of sugar. In children who are not too fat 1/4 to 1/2 teaspoon margarine or butter can be added to this cereal which is fed by spoon.

The next change in nutrition should take place in the course of the fourth or beginning of the fifth month of life and consists in the introduction of a vegetable meal. Today it is considered "modern" to start this vegetable meal much earlier, possibly already during the first three months. It has repeatedly been shown that vegetables are taken and tolerated by young infants already around that time. In general these premature and for the intestinal tract at this age not always harmless additions of vegetables seem superfluous. After the fourth or fifth month a number of vegetables can be given in puréed form, cooked with water and a trace of salt, and made tastier by the addition of roasted flour. Carrots, spinach, cauliflower, asparagus, green peas, salad and during the winter occasionally a purée of lentils, which are rich in iron, can be used. It is recommended to mix from the beginning the puréed vegetables with mashed potatoes or pre-cooked infant farina, with a relationship of two parts vegetables to one part mashed potatoes or farina. In children who have difficulty adjusting to the new taste the initial quantities given are one to two tablespoons which are increased rapidly to about 200g, that is, an average plate full. Good eaters can immediately be given the entire meal. For preparing these quantities of vegetables about 300 to 500g of raw vegetables are required. The vegetables are cooked in water to which a little salt and spices (parsley, celery, etc.) have been added. It is not necessary to cook the vegetables in meat or bone broth since the nutritional value of the meal is thereby not increased. In order to conserve the salts and vitamins contained in the vegetables it is advisable to use a pressure cooker where the vegetables are cooked rapidly and with a minimal amount of water. If the vegetables are cooked by the ordinary methods they should be steamed in very little water and not boiled. Part of the liquid, about $^1/_3$ to $^1/_4$, which is separated in the cooking process is again added to the vegetables. The vegetable meal is best given at noon, and the cereal which had previously been given at that time is offered in the late afternoon or as the last meal of the day. This schedule is made easier by the fact that the last meal is no longer given at 10 PM but already around 8 PM. The child now receives only four meals which in large, well eating children can amount to 200 to 250g.

The menu of a child between the fourth to sixth month of life would thus be as follows:

1) 6 AM: 150–200g whole milk with 5% sugar (= $1^1/_2$–2 teaspoons) and 1% butter (= $^1/_4$ teaspoon).
2) 10 AM: fruit purée made of bananas, fruit juice and sugar, 150–200g.
3) 2 PM: 1 plate mashed vegetables and potatoes.
4) 6 PM: 150–200g whole milk, without addition of water, made into cereal. 2–4 teaspoons raw fruit juice.
5) 10 PM: 150–200g whole milk with 5% sugar and 1% butter.

After the fourth month of life the $^1/_2$ or $^2/_3$ diluted milk which had hitherto been given is replaced by whole milk with sugar added, in order to meet the increasing protein requirements of the child. But still the total quantity of milk does not exceed the maximum of 500 to 600g a day. It is advisable to limit the milk intake to these quantities although far larger, almost unlimited quantities have been recommended as harmless. Around the time of the fourth to fifth month of life it is possible in many children to reduce the meals from five to four. The desire of the child should thereby be taken into account. While some children around this age gladly do without the last meal at 10 in the evening and can be awakened only with difficulties, other children will demand their evening meal beyond that age. The transition from five to four meals a day requires a change in schedule. In order to avoid the very long pause during the night the first meal of the day can be given at 6 AM, the last meal at 8 PM. The intervals between the four meals are thus increased to four and one-half to five hours. These greater intervals are of advantage because the children start their meals with better appetite and greater hunger.

The menu of a five to six months old infant thus is as follows:

1) 200–250g whole milk plus 5% sugar.
2) fruit purée of bananas, biscuits or zwieback, sugar, fruit juice.
3) mashed vegetables with potatoes or farina, 3% roasted butter or margarine plus white flour.
4) cereal made with 200–250g whole milk, 5% sugar and fruit juice (50g).

Around the sixth month of life ½ to 1 yolk of a chicken egg is mixed raw with the cereals or the vegetable. The use of raw chicken eggs seems without danger although the occasional transmission of salmonella infections through uncooked chicken eggs has been described, even though it occurs much rarer than with duck eggs which are always dangerous if given uncooked. Around the time of the ninth month of life children who are ready to eat more solid food receive for their second breakfast only some raw fruit with zwieback or biscuit. By the time the child is nine months old meat can be added to the noonday meal unless ideological considerations speak against it. Two to three times a week the child receives at noon two to three teaspoons in the beginning, later up to two tablespoons of lean meat in form of finely chopped or puréed meat or some fish given together with the vegetables. Meat and egg should be given on alternate days. Among the types of meat the organs rich in cells such as liver, brain and sweetbreads have been especially recommended since these organs because of their high purin content are said to exert a particularly favorable influence upon immunity. There is, however, no proof for this assumption. This food is prepared by cooking about 100g in salted water, then finely chopping or grinding it and frying it lightly. Around the time of the ninth month of life the child would thus eat approximately the following menu:

1) 7 AM: 200–250g whole milk with $1^{1}/_{2}$ to 2 teaspoons sugar.
2) 10 AM: Raw fruit and zwieback or biscuit or cookies and 50g whole milk.
3) 1 PM: 1 plate vegetables, mashed potatoes, meat or egg yolk.
4) 5 PM: Raw fruit and zwieback or cookies, possibly 50g whole milk.
5) 8 PM: Cereal prepared with 200–250g whole milk, fruit juice.

At the end of the first year of life children who have learned to bite and chew rougher food receive in the morning bread with butter or jam or honey with their milk. The cereal in the evening is replaced at least three to four times a week by one to two slices of bread with butter and sausage or mild cheese and raw fruit (apple, banana, etc.). Among the types of bread mothers prefer the lighter ones, without justification. There is no reason why a child of that age should not also eat dark bread. This bread which is prepared from less refined flour contains nutritive substances which are lacking in bread made from finely ground flour. For the same reason it is wrong to give the child bread without the crust. Even infants without teeth are able to chew bread crust which in the baking process develops substances which are of special nutritional value. Desserts and puddings of any kind are recommended and permitted at that time to complement and vary the diet. It is certain that the possibilities which are hereby given are in general, not fully utilized for the diet of the older infant. In addition, the white of the egg, which is preferably avoided until the child is over nine months old since it can cause symptoms of sensitization in form of eczema or strophulus (Lichen urticatus) in the young infant, is now included in the menu of the infant in cooked or baked form. The egg white can also be beaten and given in desserts or with biscuits. The addition of meat and egg during the last third of the first year of life should, however, not lead to a diet built almost entirely on these substances and large amounts of milk. This diet, which even today is occasionally still recommended as particularly fortifying, is not indicated for the healthy infant. Even at the end of the first year of life the diet of the infant must essentially still contain milk and vegetables. Only the knowledge that meat, eggs etc. contain certain substances which even in small quantities are able to further the development of the infant has resulted in the inclusion of these foods in the diet of the child below one year of age. The menu of the one year old child is essentially very similar to that of the adult. At this time the child already adjusts to the family household and the preparation of special foods is hardly necessary.

The following approximate menu is indicated for a one year old child, although far reaching variations are now possible depending on the habits and customs of the different families:

1) 200g whole milk with sugar, bread and butter and jam, tomato, fruit.
2) Raw fruit.
3) Vegetables, potatoes, meat or fish, dessert or compote, fruit.
4) 100g whole milk, bread or zwieback or cake.
5) Bread with butter and sausage or cheese or egg; fruit, maybe twice a week cereal with fruit.

The nutritional regulations which have been mentioned above, particularly for the infant during the second half of the first year of life, are only one possibility how young infants can successfully be fed on an artificial diet. This regime is recommended on the basis of the successful raising of a large number of infants on a diet similar to that outlined above. Variations and deviations from these rules are customary and permitted. Although such variations occasionally impress the layman as something new or entirely different the physician should realize that any nutritional regime is adequate for a healthy infant so long as it meets all quantitative and qualitative requirements and regularly results in the development of a healthy infant. For the individual case every physician is given considerable freedom in the composition of the menu for the healthy infant. On the other hand the individual desires of the infant should be taken into account. Children who refuse even at the end of the first year to chew solid food will remain beyond the first year of life on the pasty and liquid diet of the young infant. One must, however, take care that this type of diet also contains all the quantitatively and qualitatively necessary nutritional substances. In the same manner one should accede to the wishes of the child whether the food should be more or less sweetened or more or less warm or cool. Only in this way can the development of feeding difficulties and forced feeding be avoided (see p. 526).

ARTIFICIAL FEEDING OF THE FULLTERM NEWBORN

Because of the particular sensitivity of the newborn toward artificial nutrition every infant should, if at all possible, be naturally fed, at least during the first days of life. Only in cases where there is no secretion from the breast or where the nursing process cannot be introduced for compelling reasons artificial feeding can be started from the first day.

Today it is usually possible, in the private home as well as in the institution, to bring about under somewhat hygienic conditions a normal development of the child with cow's milk mixtures from the first day of life.

Failures in artificial nutrition of a newborn usually arise through faulty technique of artificial feeding. The first reason is undernutrition of the child leading to lack of development and diarrhea. Other authors regard the heterotrophy, the non-type-specific food, as cause for the failures. The cell ability is still limited to such an extent at this young age that any stronger claim on it, as for instance the necessity to digest and assimilate hetero-specific food, easily results in a breakdown. Perhaps difficulties in the bacterial flora in the intestinal tract might also be of significance for the inability to feed certain newborns with artificial food. Even before the first food intake bacteria from the child's surroundings enter the intestinal tract which at birth was still germ free, through mouth and rectum. The question as to which bacteria finally develop in the intestines, which flora takes possession of the intestines and becomes predominant, is determined by the first food which enters the intestinal tract. Normally a uniform bifidus flora develops under the influence of colostrum and breast milk. In cow's milk feeding there is mainly a settling of a physiological bacillus coli. The control of the intestinal flora is apparently much easier for the intestinal cells if breast milk is the medium for bacterial growth than if cow's milk constitutes the medium. In the atmosphere of the private home which is freer of germs the undisturbed establishment of the necessary symbiosis with intestinal bacteria is possible even with artificial nutrition. The massiveness and diversity, on the other hand, with which bacteria settle in the hitherto germ-free intestinal tract in children raised in institutions makes it much more difficult for the intestinal cells to establish a balance which is necessary for a physiological and useful intestinal activity, between host cells and intestinal bacteria especially in the first days of life. Not the cow's milk in itself is the cause for the failures in artificial nutrition of the newborn. The struggle against the invading intestinal bacteria occasionally leads to the catastrophy. The development of diarrhea of the newborn in institutions shows the dangers in the nutrition of young infants with hetero-specific food (see p. 428). In order to avoid these dangers the care of the newborn should be as "aseptic" as possible. This includes cleanliness of eating utensils, a careful handling of the milk and the diluting fluid and, perhaps most decisive, a high degree of cleanliness on the part of nurse and newborn. The most important factors there are special cleanliness of the hands during preparation of the food and during feeding and caring of the infant.

The customary schedule of artificial nutrition cannot be applied to this period of "extra-uterine dependency." Artificial food for the newborn "can never be prescribed on a wholesale basis but must be carefully fitted and measured to the individual" (*v.Pfaundler*). Despite the need to observe the reaction of these children particularly carefully and to determine type and quantity of food for the individual needs, it appears possible and advantageous to give some general rules as to the quantity to be chosen and the composition of the food.

The experiences with breast feeding give us a lead as to the food quantities to be chosen. Even the breast fed infant never receives right from the beginning the food quantities which correspond to his weight, which is about ½ liter breast milk with a caloric value of 300. It is therefore a mistake to give a newborn child during the first days of life immediately larger quantities of an artificial feeding mixture. Smallest quantities should be given in the beginning. After the first six to eight to 12 hours of life during which only small quantities of tea or boiled water sweetened with saccharine are given, the selected feeding mixture is first given toward the end of the first day of life, starting with five to six meals of 10cc each and increasing each meal from day to day by another 10–20cc, so that e.g., on the fourth day of life 5 × 30cc = 150cc food are given, at the end of the first week of life 5 × 60–80cc = 300 to 400cc of the feeding mixture.

If the child begins to gain weight already with these small food quantities—and this occurs not infrequently—any further increase in food should be delayed until the weight gain comes to a standstill. Because of the caution necessary in determining the food quantities it is indicated to weigh the child at a certain time of day, daily or at least twice a week, until about the end of the first week of life. As long as the food quantities are small the lack in fluid intake should be made up by small quantities of tea or water, sweetened with saccharine (about 100 to 200cc a day). These liquids should be given following the meals or in children who fall asleep immediately following the meal it can be given between meals, with the teaspoon.

During the second week of life the food quantity is increased to the point that it will meet the nutritional requirements of 100–120 calories per kilogram of body weight. In an infant with normal birth weight this quantity amounts, with the customary feeding mixtures, to 500–600cc of the milk mixture. This quantity is distributed among five meals.

In the individually cared for child, which generally drinks better than the child raised in an institution, it is possible to start the artificial nutrition of the newborn with a mixture of $^{1}/_{3}$ milk, $^{2}/_{3}$ water and 5 to 8% sugar (preferably special sugar mixtures). Equally good, and recommended for nutrition in institutions, is a mixture of $^{1}/_{2}$ milk, $^{1}/_{2}$ water and 5% to 7% sugar. During the first week of life only sugar is used for enriching the feeding mixture. After the first week of life, around the tenth to twelfth day, water should be replaced as diluting liquid by thin oat or rice water (2%). The majority of newborns thrives on this type of nutrition.

With successful artificial nutrition the physiological weight loss is rather less than with the usually slowly increasing food quantities in breast fed infants. With a steady increase in food quantities the birth weight is usually regained after 10 to 14 days. In contrast to breast fed infants the majority of artificially fed newborns is constipated. The production of few and solid stools should not give rise to far reaching changes in the composition of the food. It suffices to replace special infant sugar mixtures by cane sugar or rice water by oat water in order to improve the constipation. If we succeed in providing with artificial nutrition a start in the development of the child and getting the child beyond the first weeks of life without damage, the result of the child's further development need in no way lag behind the results obtained in successfully raised breast fed infants.

In institutions diarrheas occur occasionally which, if aggravated, develop into acute gastro-enteritis and require early and suitable treatment. The plain milk-water-sugar mixture should therefore be replaced in institutions by ½ milk—5% sugar mixtures to which an acid (lactic acid or hydrochloric acid or citric acid) has been added. This addition prevents the spoiling of the milk, the milk curds in the stomach of the infant become finer and resemble more the casein curds developed by breast milk. This supposedly facilitates digestion and absorption of milk. Buttermilk, sweetened with 5% sugar, is also a suitable food for the first period of life. It is best prepared from one of the commercially available buttermilk powders. The buttermilk which can be purchased in dairies is not recommendable because of its occasionally high acid content. Only in an emergency can the dairy buttermilk be used as infant food, after boiling it for 15 minutes and adding 1g sodium bicarbonate per liter. However after two to four weeks, provided the child develops well and eliminates normal stools, the buttermilk should be replaced by ½ milk and 5% sugar and 2% flour water. Buttermilk, because of its lack of fat, is an incomplete food for the healthy infant for a longer period of time. The fat lacking in the buttermilk can, however, be supplemented in form of 1 to 2% flour roasted in fat.

FEEDING OF THE PREMATURELY BORN INFANT

An exact definition of "prematurity" is difficult. In most cases the duration of pregnancy cannot be stated accurately. The indefiniteness in determining the time and the vagueness in establishing so-called signs of maturity in the newborns have resulted in the choice of simple indications for judging prematurity. Children with a birth weight of less than 2500g and a length at birth of less than 45 cm are considered as born prematurely. It must be taken into account, however, that even full term infants will occasionally show such low measurements. On the other hand there are prematurely born infants with normal birth weight and length. These are infants, who if carried to full term, would probably have reached a weight beyond average figures (3800 to 4000g and more) and a corresponding body length. Not all "real" prematures and not all underweight but full term infants show signs of "lack of viability." Whether a child is a true premature or a full term infant with below normal measurements can in some of the children be determined by the observation of their later development. Prematurely born infants develop stigmata in the second or third month of life which permit a ready differentiation of the prematurely born infant from the full term infant. This includes megalocephalus, large protruding eyeballs, thick tongue, short neck, short extremities etc. All these signs are lacking in the full term but small child which throughout infancy remains a well proportioned but small infant.

All premature and weak children require special care. A number of disturbances and diseases endanger all these infants with great regularity. The dangers to the prematurely born child vary in the different periods of the first year of life. These dangers are:

1) In the beginning of life—damage due to birth trauma.
2) In the first four to six weeks of life—strong lability of the intestinal tract, lability of nutritional state and immaturity of heat regulation, breathing and swallowing.
3) From the third to sixth month of life—special tendency toward the development of deficiency diseases (rickets, tetany, anemia) and very low resistance against infections.

The lower the birth weight, the lower are the chances for survival. Only with a weight of 1500 to 1800g does the prospect of keeping the child alive increase. Prematures with a birth weight below 1000g have a mortality rate of 80 to 90%, although some institutions which are specially equipped for the care of these infants, with competent nursing personnel, have reported better results even in children with a birth weight of 800g or below. The mortality rate of prematurely born infants, under favorable nursing conditions, shows the following average:

Birth weight	*Mortality*
1000–1500g	50%
1500–1800g	18%
1800g and more	7%

In individual cases it is possible to keep alive and raise newborns with a birth weight of 750-800g and less. If prematurity is accompanied by a further damage, e.g., congenital syphilis or toxemia of pregnancy of the mother, the child is doomed with almost complete certainty.

The special vulnerability of the premature was previously explained by a "lack of viability" of the child. The assumption of such a lack of viability is only justified in cases where damages originate on the part of the mother (toxemia of pregnancy, placenta praevia, preeclampsia, advanced age, etc.). The processes of digestion, metabolism and growth are, in fact, particularly intensive in premature infants since the majority of surviving infants compensate complete for their congenital handicaps in the course of one to two years. The assumption of a lower living potential which supposedly permanently weakens the premature child is therefore unjustified.

Anatomical research has shown that the previously believed but not proven assumption of debility could in many cases be explained by pathological tissue changes in vital organs which were the cause of death. The changes consist of more or less extended hemorrhages which originate in the easily rupturing blood vessels in brain, lungs, intestines, liver, kidneys and skin. The tearing of the vessels is brought about by the phenomenon occurring in every birth, that those parts of the infant's body which have already left the birth canal are exposed to a lower pressure than the parts which are still subjected to the contractions of the uterus. The drawing of blood into the parts which are under lower pressure leads to hemorrhage in immature vessel walls. The obstetrician must never be accused for the development of such hemorrhages. All stages, from petechial hemorrhages to large ecchymoses with deep tissue damage can be found. Location and extent of hemorrhages are decisive for the viability of the prematurely born child. Minor hemorrhages in the medulla oblongata can lead to death while extensive hemorrhages of the skin might disappear without leaving any damage. Particularly hemorrhages in the brain and the meninges which lead to asphyxia and to convulsions have a serious prognosis. Disease carrying germs settle in lungs and intestines in the tissues which were destroyed by the hemorrhage so that the children die of pneumonia or of a nutritional disturbance from the infection. The danger of unlimited spread of an infection is particularly great in prematures with their deficient or lacking leukocyte reaction and their inability to form antibodies. The assumption that hemorrhages in the tissues cause damage and death of many prematurely born infants has not remained uncontested. Aspiration of small amounts of food leading to aspiration pneumonia, earliest intestinal infections and other factors appeared more essential to some authors in explaining the high mortality rate of prematures than hemorrhages in vital tissues.

Beginning during the first days of life and lasting to the third month, a special sensitivity of the intestinal tract exists in prematurely born children. Disturbances in the digestive processes are accompanied or followed by a lability in the building up of the body which appears here in form of edema or weight losses, dystrophy and atrophy. The rapid and large water losses are caused in prematures by the defective ability of urine concentration although the quantities of urine as compared to the amount of water intake are lower than in the full term child. The lack of ability to economize water in the urine explains the particularly rapid withering of prematures in any disturbance.

This nutritional lability is accompanied by a further disadvantage, the incompletely developed ability of normal heat regulation which again is the result of immaturity of the nervous centers. The dangers caused by this thermolability which appears mainly in form of easily and rapidly developing undertemperatures has led to the construction of special heating apparatus, so-called incubators, and even entire heated rooms which provide for the premature infants the external heat which they themselves cannot generate internally. These incubators were unsuitable as long as heat and humidity could not be regulated. Newer apparatus which fulfills these requirements facilitates the care of the children considerably since it eliminates the constant maintenance of hot water bottles and the child lies and is cared for without clothes. Since these children do not leave the closed incubators for many weeks and all nursing care such as cleaning, weighing etc. is carried out within the closed incubator this offers an ideal means of isolation and keeps infections away from the sensitive children. The importance of avoiding primary and secondary infections is perhaps best shown in the appearance of the stools of prematures who are raised in an incubator, isolated from their surroundings. These children eliminate with great regularity once, twice or three times a day a pasty, frequently formed yellow stool. The liquid and loose stools which are so frequently found in newborns are absent in this type of nursing arrangement. This undisturbed development of intestinal functions by avoiding the invasion of a number of different bacteria contributes essentially to the successful raising of prematures in the incubator. Furthermore, appropriate installations facilitate breathing in asphyxic children or in children with a tendency to breathing difficulties, by a steady and controllable oxygen flow which, without incubator, can be accomplished only by improvised oxygen tents or direct supply through a catheter introduced in the nose.

In larger prematures light bulbs and water bottles which do not interfere with the access of light and air provide adequate protection against heat loss. Artificial heat supply must be continued until the child can by himself maintain a body temperature between 36.5 to 37. This can usually be accomplished by the time the child reaches a body weight of about 1600 to 2000g. Until that time all nursing procedures which result in a cooling off, such as baths, taking the child outdoors, must be avoided since any cooling off in prematures will rapidly interfere with the entire metabolic processes and lead to irreparable damages. The missing ultraviolet light which is of special importance for premature children with their tendency to rickets can today be replaced by the intake of appropriate vitamin preparations (see below).

Disturbances of respiration caused by the immaturity of the respiratory center are frequent. Premature children "forget" to breathe and the immature breathing centers do not react in the normal manner to the accumulation of carbon dioxide in the blood. In addition, the alveolar walls are, as a result of immaturity of the lungs, thicker than in the full term infant and at times covered with mucus which is rich in eosinophils. This mechanical obstacle also contributes to the develoment of breathing disturbances, although the hemoglobin which is characteristic for prematures, absorbs oxygen easier, even with low oxygen tension, and gives it up less freely to the tissues than the hemoglobin of the mature child.

The incomplete development of sucking and swallowing reflexes leads very easily and frequently to difficulties in food intake and to aspiration of food.

In the nutrition of premature children a number of peculiarities in metabolism must be considered. The premature child differs from the mature child with regard to digestive processes in the following points which must be taken into consideration in establishing the nutrition of the child:

1) Liver functions are not yet fully developed. Production of vitamin K is, therefore, very low, a fact which partly explains the bleeding tendency of prematures.

2) Hypoglycemia exists in the premature child which is even lower than the low blood sugar values which are seen in every young infant (about 35 mg% in prematures, 50 mg% in mature children).

3) Premature children have a hypoproteinemia, with a particularly low albumin content in the plasma.

4) Nutritional requirements of prematures are not considerably higher than those of full term infants (120 calories per kilogram of body weight as compared to 100 calories and more per kilogram of body weight in mature children). The skinniness of prematures, that is, a relatively large body surface with correspondingly great heat losses is essentially responsible for the increased nutritional requirments.

5) While digestion and absorption, particularly absorption of protein, is adequate in the premature, the ability of digesting and absorbing fat is deficient. This fact, together with the need for supplying more building material with the food because of relatively rapid growth, results in the necessity to give feeding mixtures which are rich in protein and low in fat content. Digestion and absorption of carbohydrates, on the other hand, are well developed.

Because of the undeveloped ability of fat digestion and because of the high protein requirements (up to 6g per kilogram of body weight) of prematures with a birth weight of less than 1500 g and an intra-uterine stay of less than 8 months breast milk should be replaced by artificial feeding mixtures, poor in fat, rich in protein, such as buttermilk or fat poor protein milk (half skimmed milk). Nevertheless the physician should attempt to maintain the ability of the breast to produce milk. The fulfillment of this requirement is technically not so simple since greatly underweight children cannot produce sufficient sucking stimulation. Most mothers of premature infants have no choice but to empty the breasts as far as possible by manual or mechanical expression or pumping during the first weeks of life. Some mothers prefer to express the milk by pressure on the areola while others obtain better results by emptying the breast every four hours with a manually or electrically operated breast pump. In many cases the breasts soon fail under this non-physiological process, and in other women the strong sensitivity of the breasts makes it impossible to continue the expression of milk in adequate quantities. The results are inflammations and mechanical irritations in the area of the nipple. The possibility to set and maintain in operation the breasts of a mother who gave birth to a premature and weak child by placing a strongly sucking child on her breast should be applied with caution and only in exceptional cases. The danger of transmitting a syphilitic disease from the mother to the strange child or vice versa must always be considered in such procedures. In order to facilitate the procurement of adequate quantities of breast milk many cities have set up so-called breast milk collecting stations where breast milk is sold. The price of this milk which is sold from institutions or milk collecting stations is, however, quite considerable.

The number of meals into which the daily quantum of food is divided need not exceed six in well drinking premature children. In the majority of premature children who during the first period of life are almost always bad drinkers, the number of meals must be increased to eight, 10 or 12 a day. But even in such a case the distribution of meals should as far as possible be such that there is a longer interval during the night.—There finally remains a rather considerable number of premature infants (most of those under 1500g) in whom the drinking or sucking ability is practically completely undeveloped. The method of introducing food to such children in very short intervals from a spoon which is bent together in front or through a dropper in smallest portions into nose or mouth, should be applied with great caution and restraint. Failure or absence of the swallowing mechanisms leads to entrance of food into the trachea with every drop which enters the throat and since the defense mechanism of coughing is still absent to aspiration. The regular feeding through a stomach tube which is introduced through the mouth six, eight to 10 times a day is the method of choice in these smallest and undeveloped prematures. Assuming a correct technique is used tube feeding is the best method of feeding prematures. It is, above all, the easiest way to avoid the aspiration of food which with any other type of feeding becomes the most frequent cause of death in prematures.

After introducing the stomach tube which is clamped off with a hemostat, the end of the tube is submerged in a small vessel with boiled water and the clamp is opened. If no air bubbles come up the tube is not located in the air passages. The real feeding must not be started until this test is made. The tube is clamped off again and the loose end of the tube is now connected with a small container (e.g., the glass barrel of a syringe) which holds the measured and pre-heated amount of food. By lifting the container and opening the clamp the food will slowly flow in. It sometimes takes several minutes until the infant is "ready" to accept the food. Quiet breathing and sucking motions indicate the moment. After the food is introduced—it is advisable to stir the food repeatedly during the flowing-in process in order to avoid obstruction through curds—the tube is removed. Tubes made of special material (polyethylene) can, if necessary, be left in place for 12 to 24 hours.

For the stimulation of badly drinking prematures the intramuscular injection of small quantities of cardiazol in dextrose several times daily has been recommended. In the first days of life the excess of carbon dioxide in the blood, which is frequently observed in prematures, decreases the liveliness which is necessary for the food intake. The regular supply of oxygen is not only of significance for regulating the breathing activity and eliminating cyanosis, but is also an essential stimulation for the food intake.

The determination of food quantities should be as exact as possible in the premature child. The danger of exceeding the nutritional requirements is particularly great in these children. It used to be customary to give prematures an excess of food. Today the caloric requirements of the premature child, at least during the first weeks of life, are established at about 120 calories per kilogram of body weight. There is a tendency to give rather less than this amount since these children with their minimal activities use up very little energy through muscle exercise, crying or moving about. In addition, the water requirements of these children are in the beginning lower than in fullterm infants, since they sweat less and the insensible perspiration through skin and lungs is lower than in the fullterm infants. Premature children furthermore have relatively large water reserves in their water rich tissues. The recommendation of giving these infants the minimum of their requirements decreases the danger of acute detrimental episodes which can easily occur in the premature infant during the beginning of life if the capacity of digestion and metabolism is exceeded. Experience shows that growth is possible even with these small food quantities, provided that the relatively large protein and salt requirements of the premature child are met by appropriate supplementation. Breast milk which is poor in protein is not sufficient for this. The attempt to compensate for the protein deficiency by further increasing the quantities of breast milk usually leads to failure. By adding small quantities of some protein-rich food to the basic food the child obtains the necessary protein quantities. For this purpose the addition of small amounts (5 cc in the beginning, later 10 cc and more) of sugared buttermilk is suitable, which is best prepared in the beginning not as customary with 10% but only with 5% of a buttermilk powder, with 3 to 5% of a sugar mixture added. The commercially prepared skimmed protein milk powder is also usable whereas the usual protein milk should be used only with caution in the smallest prematures because of its higher fat content. In somewhat larger prematures with a higher birth age sweetened protein milk can also be added in small quantities. Protein milk because of its low whey content has certain advantages as compared to buttermilk in children with a tendency to edema formation. Because of the still undeveloped ability in the smallest prematures to digest protein, the addition of small quantities of protein hydrolysates (amino-acids) or human plasma (which, however, must be free of erythrocytes) has been recommended for meeting the protein requirement. Amino-acid powder is added in amounts of 1–2% to the food, plasma is given in quantities of one, two or three teaspoons at every meal. Supplementing the protein requirement with buttermilk rather than adding protein hydrolysates or amino-acids has the advantage that the milk at the same time provides the necessary amounts of minerals, particularly calcium salts. Because of the particularly undeveloped ability to digest fat the fat of breast milk should in the beginning be removed through centrifugation for prematures under 1300g. The caloric deficit which thus results is to be replaced by addition of sweetened buttermilk. Only after eight to 14 days a gradual transition to whole breast milk takes place.

Forcing the premature child to gain weight during the first two weeks should be avoided. If a weight gain is forced early in immature children, the strongly marked readiness of the premature child for water retention will frequently result only in pre-edema or edema which at the first opportunity will again be lost. On the other hand, the danger of exceeding his tolerance is great. The period of nutritional lability with its danger of a severe disturbance and resulting atrophy of the child is best overcome if the organism is given time to mature. The feeding of breast milk from the bottle or by one of the above recommended special feeding methods, particularly tube feeding, is continued until the child reaches a weight of about 1500 to 1800g. Only then will the sucking ability have improved in most children to such a degree that they can drink from the bottle and later can be put on the breast.

The following summarized rules can be given as guide for the feeding of premature infants: Feeding is started about 12, 18 to 24 hours after birth. The most suitable initial food is skimmed breast milk which on the first day is given in quantities of 5 cc. five to eight times a day and then gradually increased by another 5 cc per meal. At about the end of the first week of life the skimmed breast milk should be replaced gradually by similar quantities of non-skimmed breast milk. Beginning with the fifth to eighth day of life at the latest small quantities of $^{1}/_{2}$ buttermilk (four to eight times 5 cc) or fat free protein milk or amino-acid preparations (1–2%) are added in order to enrich the breast milk with protein. If the child gains weight, even if the daily gain amounts to only 10 to 20g, a further increase is not permitted until the weight gain stops. The quantities of breast milk or protein carriers are further increased depending on the weight curve. After the initial difficulties have been overcome, the functions of sucking and swallowing developed and the child has attained a weight of about 1500–1800g the attempt can be made to let the child drink from the bottle or for a short time (five minutes) from the mother's breast. The success of this experiment, which is determined by the scale, decides whether the child can change over to normal feeding on the breast with a reduced number of meals or, in case the first nursing attempts fail, whether the previous method of feeding with a tube or with a bottle should still be continued for a while. If breast milk is not available, the feeding of eight times 5 cc $^{1}/_{2}$ buttermilk plus 5% sugar mixture or fat-free protein milk plus 5% sugar mixture should be started with at least the same caution. The $^{1}/_{2}$ buttermilk can gradually be replaced by whole buttermilk after eight to 10 days. After the fourth to sixth week and even later in the smallest prematures, the buttermilk or protein milk is no longer diluted with water but with 1% and later 2% rice water.*

If both buttermilk and protein milk are unavailable, $^{1}/_{2}$ skimmed lactic acid milk or $^{1}/_{2}$ citric acid milk can be tried.

If the dangers of the first critical period of life have been successfully overcome with either natural or artificial nutrition, the regulation of nutrition in the following months of life must be carefully controlled—in the premature child much more so than in the full term infant. The incomplete development of a number of tissues and functions, the lack of reserves of some vital substances which accumulate in the full term infant during the last weeks of pregnancy (calcium, iron) are in the immature child potential causes for the development of nutritional deficiencies of which rickets, tetany, anemia and lack of immunity are the most important. If these diseases are to be avoided early supplementation of the milk is necessary. The time at which this supplementation should begin is determined by the periods during which these deficiency diseases develop. For rickets and tetany in the premature child this is already between the first to third month of life. Transition from a physiological anemia of the premature (see p. 311) into a macrocytic, at times hypochromic anemia occurs in the second to third month of life. Already in the second to third month, but particularly in the fifth to sixth month, the lack of immunity reaches a degree which endangers the premature child.

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* A buttermilk prepared from 12% of a customary commercial preparation (instead of 10% buttermilk) is a simple method of enriching the food with protein.

The above data indicate that supplementation of food should be started by the end of the first month of life at the latest. In the third to fourth month of life the food must already be complete to a degree which for the nutrition of the full term child becomes necessary only at a later age. The need for early supplementation of the diet in various directions is important because the lack of physical development, the smallness and thinness of some prematures even in the second quarter of life creates the impression that it might be dangerous to add cereal, fruit, zwieback, vegetables, vitamins, etc. to the diet of these underweight infants. Weight and length do not determine the necessary supplementation of food. Physicians who are experienced in the care of premature infants recommend the addition of vitamins A, D and C in form of preparations containing all these vitamins, already in the second or third week of life. This early intake of vitamin preparations in suitable form (e.g. vitamin D in a *non*-oily solution) is perhaps useful. Whether it is really essential to begin at such an early age has not been finally established. The possibility has been taken into consideration that the development of serious changes in the eye (retrolental fibroplasia) which occurs in 60% of all prematures and in 15% results in blindness, may be connected with the steady oxygen supply or with the early intake of vitamin A and D.* Edema of the papilla, which develops on a diet rich in whey and which damages the optic nerve has also been considered. It is assumed that the undeveloped kidney function promotes this tendency for edema. The possibility of a connection between damage to the eyes and early vitamin intake cannot be immediately rejected with certainty. It therefore appears preferable to somewhat delay the beginning of vitamin intake, particularly since this short delay does not, apparently, impair the development of the premature.

The addition of vitamin C, 25–75 mg daily per injection or in tablet or powder form, begins in the third to fourth week of life. In place of vitamin C injections or tablets raw fruit juices (10 to 15 cc twice a day) can be given beginning about the second month of life, provided the child's development is satisfactory and there is no diarrhea.

In order to combat rickets and the occasionally accompanying tetany which are almost unavoidable in prematures, the customary prophylactic measures should be taken systematically and at an early age. The utilization of natural sun rays in form of sunbaths is out of question because of the danger of chilling. It is preferable for children with a weight of 1600–2000g to apply irradiation with an ultraviolet lamp beginning after the first or second month of life, whereby care must be taken to provide adequate warmth during the time of irradiation. These irradiations with ultraviolet rays should be continued with short interruptions, particularly during the winter months.

The addition of vitamin D for prophylaxis against rickets and tetany (see corresponding chapter p. 276) begins at the end of the first or beginning of the second month of life. Water soluble vitamin D preparations are preferable to oil soluble preparations in prematures. A daily intake of 1000–2000 units suffices (beginning with one to two drops of the usual preparations). A single large dose of vitamin D (300,000 units) has been cautioned against in prematures because of the danger of kidney damages. Apart from vitamin D, breast milk fed prematures require an addition of calcium. If buttermilk, protein milk or some other cow's milk mixture was given for supplementing the diet, the quantities of calcium salts contained in the additional food are sufficient for the rapidly progressing post partum mineralization of the bones of premature children.

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* The rapid transition from a high oxygen concentration (40–60%) in the incubator to the normal concentration (20–25%) is considered the main cause of retrolental fibroplasia. It is possible to cure this disease in its initial phase by returning the child to the high oxygen concentration for about a week. Thus oxygen is probably a factor in the development of this eye defect in prematures as it exerts an unfavorable influence on the early adaptation of an immature organ. From a practical point of view it seems important to keep premature infants in as low an oxygen atmosphere as possible (25–30%) except in case of asphyxia.

Because of the strong disposition of the premature to rickets however, a complete suppression of this disease cannot always be accomplished despite adequate prophylaxis.

Beginning with the third month of life prematures who have been entirely or to a large extent artificially fed and who develop satisfactorily and attained a weight of 1800 to 2000g can have one of the meals replaced by cereal prepared with whole milk, a second meal by mashed bananas or mashed zwieback or biscuits. In anemic children the addition of small quantities (one to two teaspoons) of puréed, cooked liver is recommended already around this time of life. From the fourth month another meal is to be replaced by vegetable and potato purée.

Considerable difficulties arise frequently in the practical execution of this nutrition. Resistance on the part of relatives who object to giving "delicate infants" such a "strong, heavy diet" can be overcome. Much more serious is the persistence with which especially premature children resist the intake of food with pasty or crumbly consistency. With progressing age, particularly in the second half of the first year of life, a large number of premature children—and twins behave similarly—develop a violent resistance against the intake of any solid food. Already in the third quarter of the first year the tendency to attacks of defiance and rage is one of the characteristics of the premature and this state of excitement also develops when the intake of vegetables or cereals is requested. Only by infinite patience is it possible to gradually overcome this period of food rejection. Even at the end of infancy and during early childhood a not inconsiderable part of the children lacking in appetite and eating badly are prematures. By applying force, which only increases the resistance and anger of the child, no success can be expected for overcoming the lack of eating desire, even in the premature child. A certain amount of yielding gives better results. It may, however, never be carried to the degree that the necessary supplementation of the diet is completely omitted, that is, that the child is fed only on milk mixtures up to the end of the first year. In every case the addition of raw fruit juices, egg yolk and anti-rachitic preparations is esential. The possibility to supply the necessary vitamins (A, B, C and D) in form of injections or tablets facilitates today the overcoming of these difficulties. Vegetables can at times still be given if they are further diluted with farina and given through the bottle. The intake of crumbly zwieback, biscuits or bread, which every time causes gagging and vomiting must, however, at times be omitted.

A complete, protein-rich diet is at the same time the only way to raise the immunity which in prematures is always insufficient and incomplete and to bring it to a level where the child is able to overcome simple infections. This applies particularly to the time beyond the third or fourth month of life. The failure of resistance against the first infection is the more unexpected and surprising since it occurs not infrequently in children whose physical development apparently was completely normal up to the day of infection. Even with optimal weight gain, excellent appearance and despite the development of normal adipose tissues etc. the premature child can be considered eutrophic only after it has successfully overcome the first infection. Only a complete diet, together with progressing maturation of tissues in the second half of the first year, develops in the premature the forces of resistance which are necessary for overcoming the unavoidable infections.

For the treatment of the physiological anemias of prematures (see p. 311) iron preparation are not very effective. They must be used with caution in order to avoid disturbances in the appetite and development of diarrhea and vomiting which occur with many iron preparations. The number of preparations which are free from these disturbing characteristics is small. Anemia can best be treated by single or repeated intravenous transfusions of 60–80cc of blood which in female prematures must be Rh-negative. If in badly eating prematures the physiological anemia has developed into the hypochromic late anemia iron and liver preparations and B 12 are indicated (see chapter on Anemia p. 319).

Hormone therapy (female or male sex hormone) which has been recommended for promoting cell maturation has not brought any definite results. This therapy can be omitted. On the other hand it seems indicated to give all prematures vitamin K injections on the first and second days of life, in order to decrease the bleeding tendency which is probably a result of defective vitamin K formation in the intestinal tract or of lack of development of liver functions.

Sclerema of prematures seems to improve with adequate heat provision together with intake of vitamin E (Tokopherol). Vitamin E is given for two to four days (no longer) in doses of 50 mg daily per os.

PART II

Pathology of Infant Nutrition

CHAPTER IV

Characterization of the Nutritional State

THE UNDERSTANDING of the nutritional state has contributed most to the progress of our knowledge of pathology and therapy of nutritional disturbances. If an adult or an older child reduces his body weight, his functional forces remain unaffected except for extreme cases. This is entirely different in the infant who is dependent on steady weight gain. With a more or less extensive consumption of his body substance materials are lost which are essential for his development and maintenance. Any interruption in weight gain or loss of weight is connected with a reduction of the entire functional capacity.

Changes to the worse from normal development and a state of health as described in the preceding pages are not infrequent during infancy. The term "eutrophia" can be used to describe the state of health. Deterioration of the nutritional state to a smaller or greater degree leads to "dystrophy." Serious damages of the nutritional state are described as "atrophy." The limits between eutrophic, dystrophic and atrophic conditions are not always clearly defined.

Any deterioration of the nutritional state of the infant is characterized by two factors. First, it is remarkable that an unimportant cause may suffice to bring about a damage of the nutritional state. Small, seemingly insignificant faults in nutrition harm the general condition of the child and his development during this period of life where the dependence on food is so much greater than in older children or adults. Secondly and much more significant is the deterioration of all functions of the organism which accompanies any deterioration in the nutritional state. Any disturbance in the development of the infant, therefore, results in difficulties in feeding, overcoming infections and in the care of the child. The determination of the nutritional state is the first task of the physician who undertakes the treatment of an infant.

The classification of a child into a dystrophic or atrophic patient is merely the description of a pathological state which can develop from many causes. By taking an exact anamnesis, obtaining a description of the course of the disease, by questions regarding the nutritional methods used, by searching for specific symptoms etc. it is possible in the majority of children to form an opinion as to the causes which led to the deterioration of the nutritional state. Only on the basis of the knowledge of the etiology is it possible to give orders regarding therapy, to give information regarding the prognosis etc. In many patients, particularly severely dystrophic or atrophic children, the etiology of the damages is frequently so varied that it is not always possible to recognize whether the causes for the deterioration of the nutritional state were faults in nutrition, infections, or faulty care of the child.

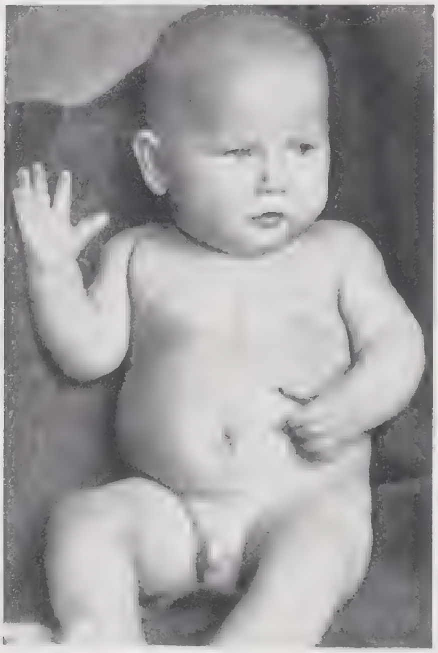


Fig. 12. Eutrophic infant.

The separation of the concepts of nutritional state and nutritional disturbance will facilitate for the physician the understanding of the complicated processes. A therapy for dystrophy or atrophy in itself does not exist. Successful treatment of dystrophy

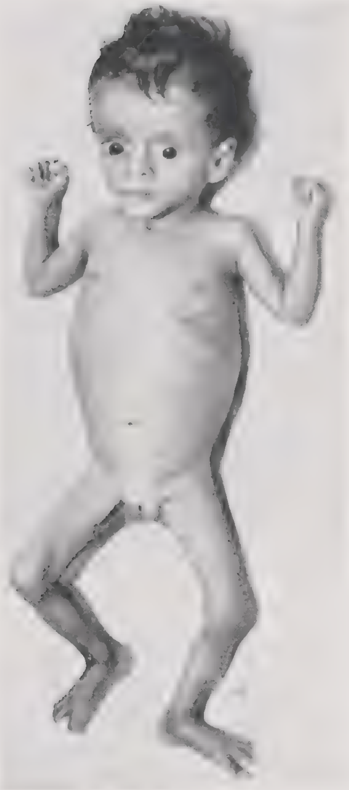


Fig. 13. Dystrophic infant.

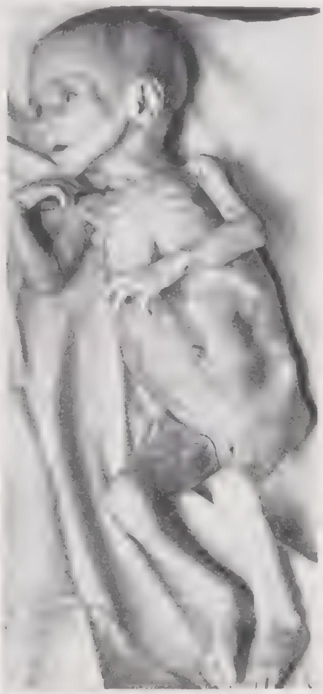


Fig. 14. Atrophic infant.

or atrophy, that is, returning the child to an eutrophic condition, consists in removal and treatment of the numerous causes leading to dystrophy or atrophy.

The disturbances which cause similar damages in the nutritional states of eutrophic, dystrophic and atrophic children or which maintain the child in a low nutritional state can be summarized in the following four groups:

1) Faulty Nutrition.
2) Diarrhea.
3) Infections, and
4) Abnormal Constitution.

CLINICAL PICTURE OF PATHOLOGICAL NUTRITIONAL STATES

The most striking sign of a deterioration of the nutritional state is emaciation or loss of weight of the child. The adipose tissues which lend roundness to the body of the healthy infant diminish or disappear. The decrease in adipose tissue alone permits an immediate differentiation of the various phases of the lowered nutritional state. The decrease in adipose tissues characterizes a low degree of deterioration while the absence of fat marks a more serious degree of decline in the nutritional state. Lesser changes are found in children who are considered dystrophic. Serious changes occur in children who are described as atrophic.

The state of good health, of eutrophia, never changes suddenly into atrophy. Between eutrophic and atrophic conditions there is invariably a period of time during which the child is in a state of dystrophy. In the newborn or the young infant, for instance, this dystrophic phase can be very brief. The younger the infant the more rapid is the transition from eutrophy to atrophy. During the first and second months of life a few days suffice at times to bring about serious damages in the infant's organism. Furthermore, the damages which cause atrophy in children of that age can be conspicuously small. In the older infant more serious and longer lasting damages are necessary to cause a deterioration in the child's condition from dystrophy to atrophy. Apart from age and the severity of the damage, constitutional factors are of significance for the speed with which the decline in the nutritional state takes place. After the second half of the first year of life only children with special dispositions will reach a state of atrophy.

The disappearance of adipose tissues to the point of atrophy does not happen in an haphazard way. Upon examination of the sick infant the study of the adipose tissues which are found to be still present or have already disappeared permits an evaluation of the nutritional state. The adipose tissues which are present in every healthy infant after the sixth to eighth week of life (see p. 32) disappear in the following sequence in case of damage to the infant's organism: First the abdominal fat is lost. Fat on chest and shoulders follows. Only then will the fat loss spread to arms, legs and buttocks. At the time when the infant has already reached a considerable degree of skinniness of trunk and extremities the face still maintains approximately its round appearance. Only a renewed or chronic damage will cause the disappearance of the highly resistant facial fat of the infant. The last stage in the fat loss, during which the fat in the cheeks and even the so-called Bichat's fat pad disappear, characterizes the state of atrophy. The disappearance of the fat on trunk and extremities with maintenance of facial fat permits a diagnosis of dystrophy. Depending on the degree and the variable extent of fat loss milder and more serious stages of dystrophy can be differentiated. The senile features which have been compared to the face of old Voltaire characterize the atrophic child. The variable speed of disappearance of the individual adipose tissues can be explained by chemical differences in the composition of individual fat deposits (see p. 35).

The disappearance of fat is paralleled by a decrease of other tissues, particularly of the musculature which in the atrophic child is finally reduced to a minimum. The losses of body substance which the infant can bear and which still permit continuation of life are considerable. According to the well known and essentially applicable figures by *Quest* a loss of more than one third of the body weight is incompatible with life of an infant. These figures correspond to statements (*Rubner*) that the infant can lose all glycogen, all fat and up to 21% of his body fluid and still live and recover. A gradual loss in body substance is better tolerated than sudden great losses whereby the organism is unable to adjust with sufficient speed.

The effect of dystrophy upon inner organs becomes apparent mainly in the decrease of thymus and ovaries, and to a lesser extent in the reduction of the adrenals and testicles. The thyroid gland, on the other hand, is almost overweight as compared to the body weight.

Decrease in weight does not signify loss in fluids, as shown by analyses of different organs and of the entire body of atrophic children. Dystrophy and atrophy are therefore not accompanied by a "drying up," provided there is no acute diarrhea. Accordingly, the blood shows only a moderate hypoproteinemia (about 4.5–5%). The relationship of N-containing substances to water is not essentially changed, even in the most severe stages of atrophy, in fact it might even be elevated in favor of the water, despite the apparent dryness or drying up of tissues as compared to the norm. Protein loss is therefore greater than would correspond to actual weight loss. The dystrophic and atrophic organism is "waterlogged." This results in a relative increase of extracellular fluid, while intra-cellular fluid is lost in relatively large quantities with the reduction of the musculature.

Not only quantitative but also qualitative changes in the fluid content of the cells take place during deterioration from eutrophy into atrophy. The turgor of healthy tissue disappears more and more with the transition from eutrophy to atrophy. The dystrophic individual already shows a loss in the elasticity of the tissues. The atrophic individual possesses only completely inelastic tissues, without form and shape. In drawing a comparison with the plant world one could say that the eutrophic individual resembles a blooming plant, the dystrophic individual a shriveling and the atrophic individual a withered plant.

The initially limited and later on completely lost ability of the tissues to grow is shown in the changes in body weight. In the dystrophic infant the weight gain lags behind the normal incline. In severe stages of dystrophy there is no weight gain at all. Children remain for days and weeks at the same weight level. A greater degree of decrease of the weight curve below the previously attained level signifies a progressive deterioration of dystrophy in the direction of atrophy. Rapid weight loss, a decrease of several hundred grams in 24 hours, occurs in the severest stages of atrophy and usually is the expression of a change to the worse. These last stages of atrophy which are accompanied by diarrhea and frequently by uncontrollable, bile-colored or coffeeground-like vomiting correspond to the picture which has been described by *Finkelstein* as decomposition.

Together with the decrease of the ability of the tissues to grow other signs of health disappear in the child. The rosy color of the skin becomes pale. The initial pallor which at first can perhaps be clearly observed only in the sleeping child, changes with any decline in the nutritional state into a greyish-yellow or brownish to livid discoloration of the skin. Simultaneously the transparency of the skin is altered. The skin of the dystrophic or atrophic child appears more transparent so long as there are no acute disturbances. The peculiarity of the skin of these children can to a certain extent be compared to the appearance of strongly diluted milk. These transformations are probably due to disturbances in circulation, changes in pigmentation and colloidal changes of cell protein and alterations in the relationship of extra- and intracellular fluid.

In contrast to the pallor of the skin the mucous membranes are strongly reddened. Already in the pale dystrophic individual the red lips are striking. The highest degree of hyperemia of the mucous membranes is reached in the atrophic child whose bright red lip and oral mucous membranes are in sharp contrast to the bluish pallor of the face. Only shortly before death will the scarlet discoloration of the oral mucous membranes become suffused with livid hues.

The tonus of the musculature is frequently elevated in the beginning of the disease, and subsequently rather diminished. In the severest stages of atrophy it is not infrequently reduced to complete atony. The paperthin, atonic abdominal wall yields to the pressure of the distended loops of gut. Disturbances in circulation, stasis in the veins of the mesentery and the intestines, accumulations of large quantities of blood in the liver which is enlarged, promote the development of meteorism, directly and through disturbances of absorption of intestinal gases; this can progress to a stage where palpation of the abdomen becomes difficult. Disturbances of the potassium metabolism are perhaps the basic cause for this hypotony of the musculature. The motions of the intestines become clearly visible through the thin abdominal walls. Another sign of atony of the musculature is the failure of the anus to close which frequently leads to a prolapse of the intestinal mucosa.

The decline in the nutritional state of the infant is accompanied by changes in the emotional behaviour of the children which are frequently noticed by the mothers as first signs of disease. The observation of the psychological behaviour gives the physician a valuable clue for the prognosis of the condition. These sick children lose their equanimity which characterizes the healthy infant who generally is content and friendly and expresses his displeasure only for a reason (hunger, uncomfortable position, etc.) by crying. The child with progressive decline in the nutritional state becomes irritable and his mood is changeable. Depression and apathy lasting for several hours may be followed by periods of restlessness and prolonged crying for which no cause can be found. The state of dystrophy is characterized by tiredness and lassitude. All pleasant feelings are lost. With a deterioration of the nutritional state to the point of atrophy the exaggerated quiet is replaced by unrest and crying, progressing to stages of excitement to which hypovitaminosis (lack of vitamin B—nicotinic acid) is a contributory factor. The changes in the emotional pattern also become apparent in the expression of the child. The eyes of the dystrophic child are dull. It appears difficult for the child to raise the lids. The atrophic child lies sleepless, day and night for hours, with wide open, anxiously searching eyes which do not rest on an object for any length of time. The irritation of the voluntary musculature of the eye-ball corresponds in the atrophic child to the motoric unrest which characterizes the total behaviour of seriously damaged children with their restless, aimless movements, with the hands searching restlessly and lying quietly for only short periods of time when between the eagerly sucking lips of the child.

SIGNIFICANCE OF THE PATHOLOGICAL CHANGES OF THE NUTRITIONAL STATE

It is important for the treatment to recognize the factors which cause a hitherto healthy child to descend to dystrophic and atrophic conditions. Different causes leading to dystrophy or to atrophy require different therapies. Dystrophy caused by quantitative hunger is to be treated differently from a similarly appearing dystrophy resulting from chronic diarrhea. Dystrophy brought about by frequent infections requires a different treatment from a dystrophy developed in a qualitatively undernourished child. It follows that there is no method of treatment suitable for every case of dystrophy or atrophy. Only by removing the damages, varying from case to case, which caused the deterioration of the nutritional state, is it possible to return the child to a state of health, of eutrophia.

Hunger of any kind and infections are the most frequent causes for the development of a decline in the nutritional state. The pathogenesis of most dystrophies or atrophies can be traced back to this simple formula. All earlier attempts to interpret these phenomena, such as the assumption of primary intestinal atrophy or a constitutional inability of the child to feed, etc. have not been proven correct. The physician must reject such excuses for explaining failures in the nutrition of his patients.

The extraordinary reaction to hunger during infancy clearly demonstrates the differences in the physiology of the first year of life. At this age hunger is not only the more or less complete lack of all substances providing materials for generating heat or for growth, even partial hunger is of similar significance during infancy. This form of hunger develops when the caloric requirements of the organism are met by the food intake, but when the food is incomplete due to insufficient supply of one or more of the heat generating or growth promoting substances (protein, fat, carbohydrate, water) or due to inadequate supply of supplementary substances (vitamins).

Apart from external hunger, light or serious, repeated or prolonged infections can become the cause for the development of dystrophy or atrophy. Usually we find here too an inadequate nutrition of the child, which might be caused by an insufficient food intake of the ill and feverish child or by an accompanying diarrhea leading to "internal hunger". The assumption that there might be specific dystrophies for certain diseases which have been considered particularly for lues and tuberculosis, has not been proven for infants. It is contradicted by the successes and the good development of luetic children who are adequately treated and nursed on the breast of their own mothers, and by the nutritional successes with tuberculous infants, provided they are free from secondary infections.

A third possibility, which is almost unknown during later childhood and during adulthood, may lead to hunger in the infant: this occurs when the supply of food is adequate regarding quantity and composition, but when the ingested food is then improperly metabolized on its way to the places of utilization, through disturbances in the activities of the gastrointestinal tract. This results in a state of "internal hunger" which has a similar effect on the cells as "external hunger" with a quantitatively or qualitatively inadequate food intake.

The nutritional state of the infant can be affected by hunger in the following different manners:

- a) Hunger due to *external* causes
    - 1) quantitatively inadequate intake
    - 2) qualitatively inadequate intake
- b) Hunger due to *internal* causes
    - 1) pathological digestion of food in the intestines
    - 2) impairment of absorption or, more rarely,
    - 3) impairment of assimilation of food which is adequate in quality or quantity.

One of these forms of hunger can almost always be found in the history of a dystrophic or atrophic infant. It is thus possible that a dystrophic condition can develop similarly in a breast fed child which does not drink sufficiently; in a child with pylorospasm which vomits a greater part of his food; and in a child whose food contains insufficient quantities of carbohydrates, protein or fat. Dystrophy may result also if diarrhea prevents regular digestion and absorption of food from the intestinal tract or if constitutionally abnormal cells make normal building up of tissues impossible. Individual differences are no doubt important for the development of dystrophy or atrophy. Some infants can be subjected to severe undernutrition for a long period of time without developing symptoms of dystrophy or atrophy.

Hunger due to internal or external causes is the greatest enemy of the infant. By avoiding hunger and undernutrition the state of eutrophia can best be maintained. A quantitatively or qualitatively inadequate diet should be ordered by the physician only upon serious consideration. The transition from eutrophy to dystrophy and atrophy certainly proceeds much more rapidly than the return from dystrophy to eutrophy. The way of recovery from atrophy to a state of health is always long and arduous.

A diagnosis of eutrophy, dystrophy or atrophy represents an evaluation of the working capacity of the organism.

Not a single function in the organism of the dystrophic and atrophic child remains unaffected by the damage. The damage to the following three functions is, however, of prime importance: 1) A child in a bad nutritional state loses his ability to fight off infections. 2) The ability of the intestines to adequately digest food and the ability of the cells to normally assimilate the food is impaired and this causes 3) a damage in the solidity of the structure of the organism.

In fighting off infections—mainly the frequently occurring grippal and suppurative infections—the ability to rapidly overcome the disease is lost. The pathological pattern in the common grippal infection is monotonous in the healthy child. Complications and "interesting" cases are rare. With a reduction in the ability to fight off the disease the aspect of the disease changes. A cold with fever and a pharyngitis which a healthy child overcomes in three to four days, develops in the dystrophic child into a prolonged bronchitis with fever or into bronchopneumonia. The atrophic individual frequently can no longer overcome infections at all. For days and weeks one wave of fever follows another. The course of the disease shows many complications. The behaviour of a child in fighting off infections is surely the most accurate way of judging the seriousness of the nutritional state, more accurate even than the course of the weight curve and other signs of health.

The severe secondary diseases which accompany and follow influenza (empyema, osteomyelitis, pyuria of hematogenous origin, etc.) occur more frequently in dystrophic infants, and are hardly ever survived by atrophic individuals. It has thus come about that the pathology of infancy has become scanty and monotonous in countries where the majority of children are well developed. If the digestive processes are affected by infections a further reduction in the nutritional state will result.

In the recovery from dystrophy and atrophy the weight gain and the formation of new tissue, although initially not of good quality, precedes the return of normal functions. The face of the atrophic child becomes already rounder, the dystrophic infant appears to accumulate again fat reserves, a weight gain which had been absent for a long time again takes place, and considerable progress seems to have been accomplished on the way to recovery. If the child now suffers a grippal infection, the manner in which this test is overcome shows whether the changes in the organism actually constituted improvement and recovery or whether it was only an apparent improvement. The failure to fight infection can come as a surprise to parents and physicians even after three or

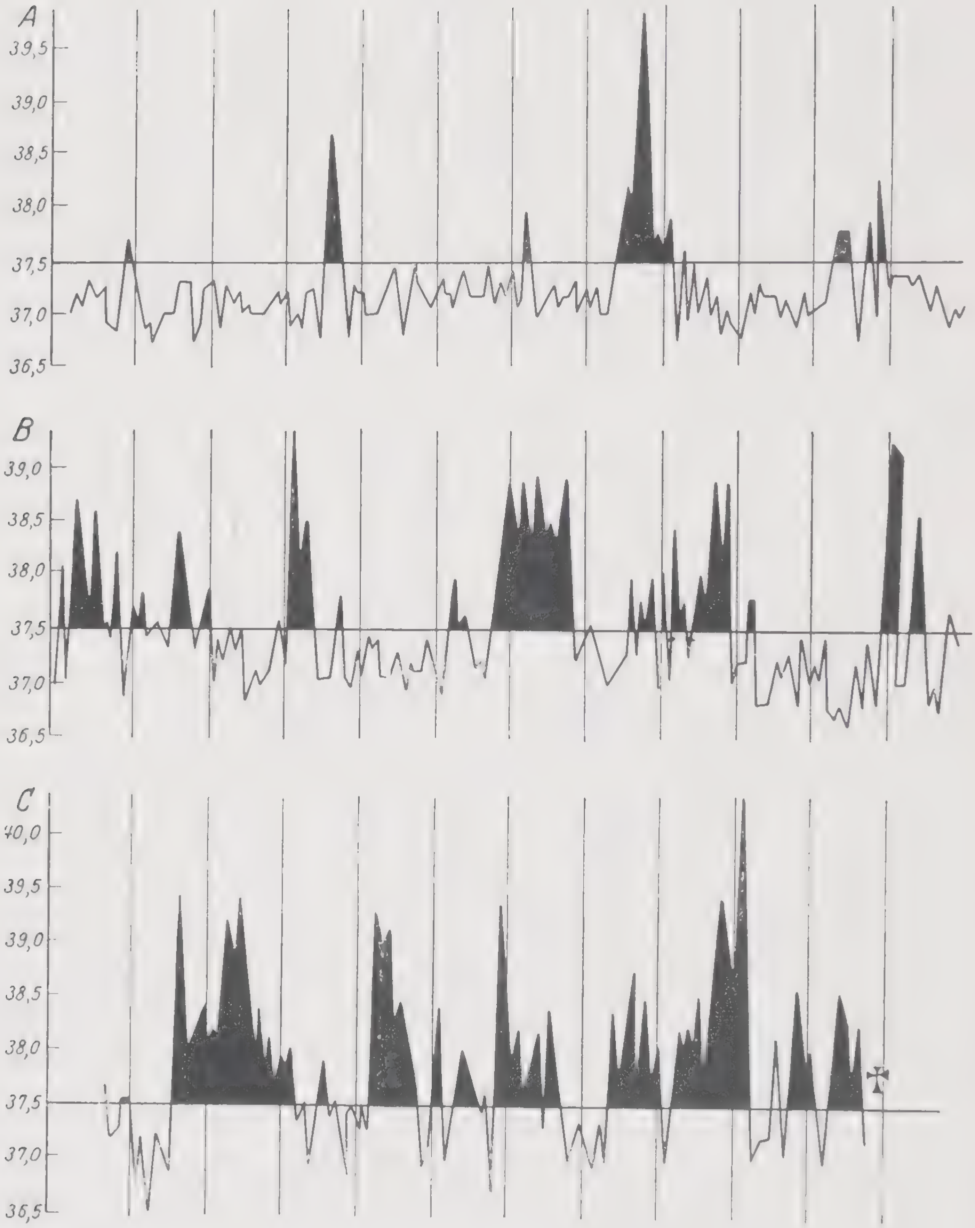


Fig. 15. Duration and height of fever and the number of infections reflect the degree of impairment of the nutritional state. A–Eutrophia, B–Dystrophy, and C–Atrophy.

four months during which the child apparently developed well and had shown signs of health.

The deterioration of the organic functions becomes most obvious in the reaction toward a renewed period of hunger lasting only hours or days. The eutrophic child tolerates the deprivation of protein, carbohydrates and fat for 24 to 48 hours without any essential change in his general condition. In a number of children there is hardly an interruption in weight gain or only a slight lowering of the weight curve. Other healthy infants lose about 100 to 200g during the hours of hunger which are rapidly replaced as soon as the child again receives food. The dystrophic child already suffers considerably if exposed to hunger for 24 hours. A longer period of hunger results in serious danger. The last fat reserves are utilized, the weight loss is considerable. The dystrophic child, particularly in the first half year of life, can turn into an atrophic individual as a result of a single day of hunger. For the atrophic infant any *prolonged lack of food* represents a *grave danger.* The temperature regulation suffers. Subnormal temperatures and a state of collapse develop already as a result of six, eight to 12 hours of hunger. The weight curve drops abruptly.

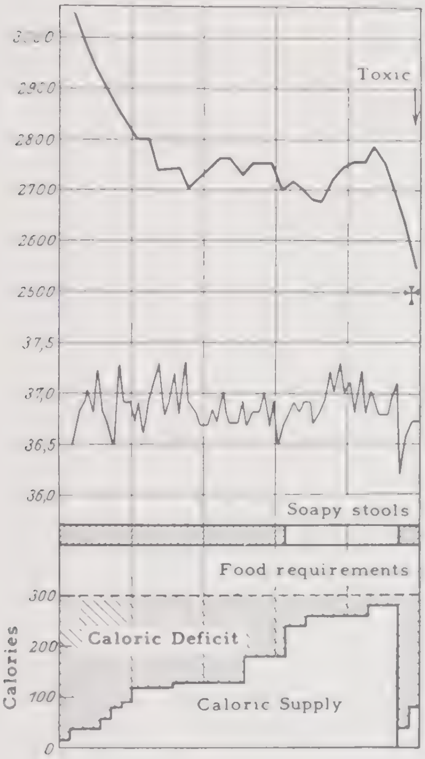


Fig. 16. Observation dating back some years. Results of unduly prolonged hunger (see caloric deficit in graph): first dystrophy, then atrophy and finally death due to infection under toxic manifestations.

But the reactions of children toward food intake also vary according to their nutritional state. The healthy child, as has been mentioned before, possesses an extraordinarily great tolerance regarding quantity and composition of food. Only gross mistakes

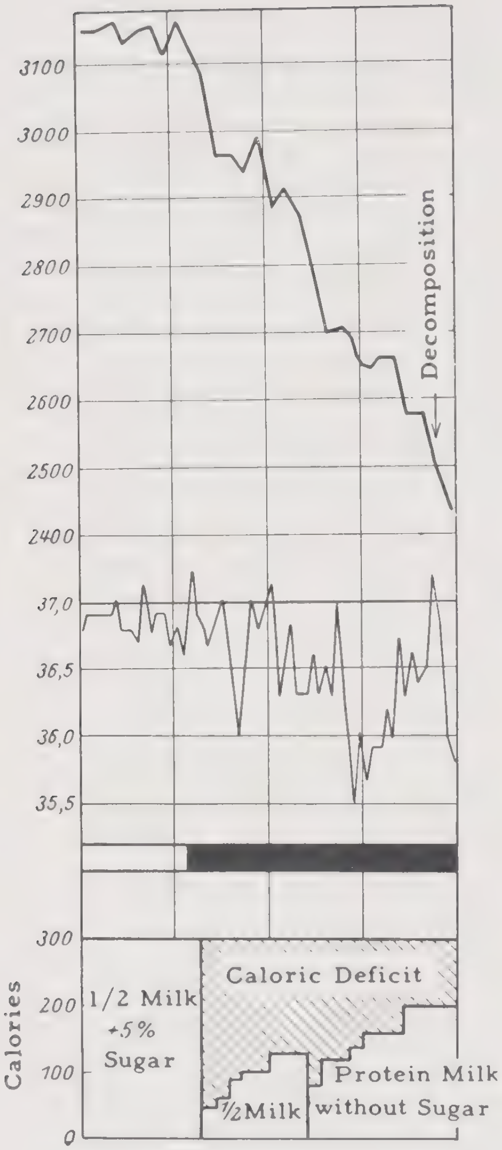


Fig. 17. Because of light diarrheas, ½ milk without sugar is given in small quantities. The subsequent loss and the increased diarrhea misled to renewed dietary changes and reduction of food quantities (now protein milk without (!!) sugar). Result: progressive deterioration of the nutritional state, beginning of decomposition with markedly low temperatures, slowing of pulse (pulse about 84) and finally death. Fatal decomposition due to hunger, especially due to lack of carbohydrates.

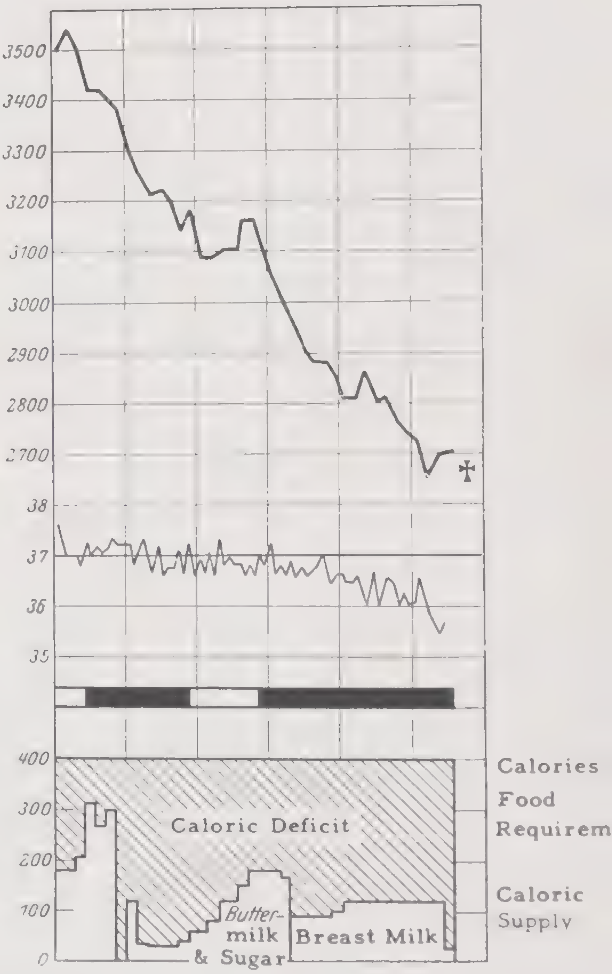


Fig. 18. Catastrophic consequences to unc nutrition. Not even one third of the nutritic requirements were covered throughout a pe of six weeks.

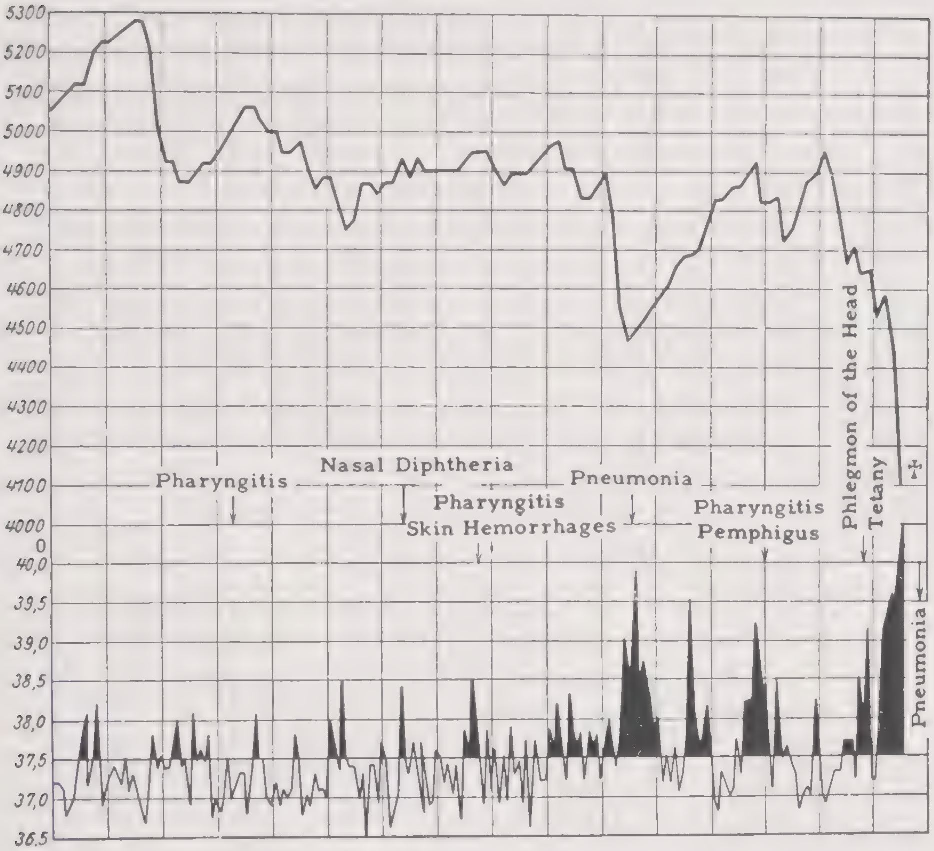


Fig. 19. Increasing number of infections (dysergia). Result: increasing dystrophy and finally death due to pneumonia.

in measuring and choosing the food lead to pathological symptoms in the healthy child. An increase in food intake results in a weight gain in accordance with physiological laws. In the dystrophic infants, however, the functionally damaged tissues have lost the ability to cope with an even moderate excess of food without harm. The pathological cells of the atrophic infant react to excessive food intake with diarrhea, weight decrease or abrupt weight losses. This has been referred to as paradox reaction.

In patients who fail to develop for a longer period of time for undeterminable reasons, their reaction to a careful increase in food quantity or to a period of hunger permits an evaluation regarding the still existing working capacity or the already developed functional damages of the cells. In the practical treatment of nutritional disturbances the careful increase of food is pref-

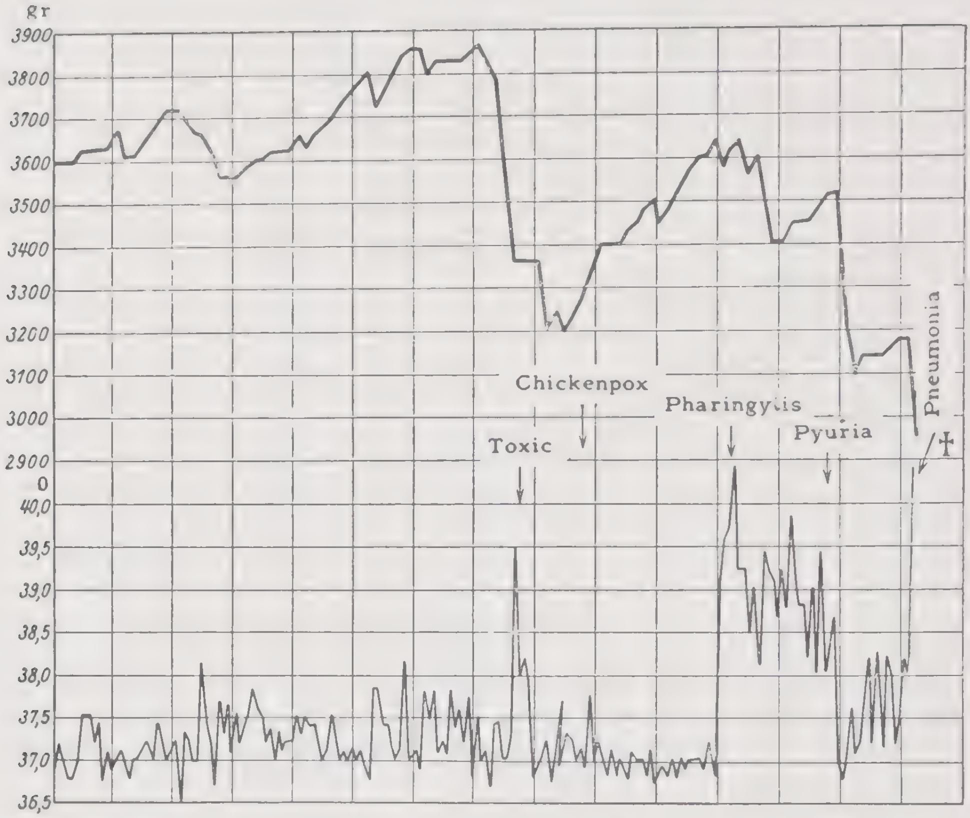


Fig. 20. Progressive dystrophy due to repeated infections which together with increasing dystrophy and dysergia led to death due to pneumonia.

erable as test for judging the nutritional state. It is always better in infants who fail to develop to force a clarification of the situation by such a tolerance test rather than have the children spend days and weeks without weight gain. Any prolonged absence of weight increase signifies for the infant progressing dystrophy.

The third functional disturbance which occurs with any deterioration in the nutritional state concerns the solidity of the tissue structure whose resistance goes down with each progressive reduction in the nutritional state. But even during the period of recovery from dystrophy or atrophy the composition of the tissues does not at first possess the solidity of normal protoplasm. The newly built tissue is labile and collapses from any minor cause. This nutritional lability is evidenced in renewed weight losses which frequently are greater than the previously attained gain.

The solidity which characterizes the body tissue of the eutrophic child is acquired only very gradually by the new tissues.

A diagnosis of the nutritional state brings important conclusions concerning treatment:

1) The prognosis of any nutritional disturbance and any infectious disease depends during infancy considerably on the nutritional state of the child.

2) Progress in the treatment of a nutritional disturbance is essentially determined by the nutritional state of the infant.

In the eutrophic child a certain amount of optimism is always permissible in the prognosis even if the disease appears serious at first. The physician has far-reaching freedom in ordering therapy.

With a reduction of food tolerance and of immunological defense, treatment and prognosis change in the dystrophic individual. It is hardly possible to predict from which side the dystrophic child is more endangered—from decreased defense against infections, from diarrhea or from the absence of stabile tissue formation. All these dangers are interlocked like the links of a chain where it is impossible to distinguish beginning and end.

Treatment of a case of acute diarrhea is more difficult in the dystrophic than in the eutrophic child. For nutritional therapy a special therapeutic diet should be given which takes into account not only the decreased tolerance but which also supports in its qualitative composition the restoration of normal intestinal functions (see later). Any delay in the treatment of diarrhea is equivalent to a further deterioration of the nutritional state. In a state of dystrophy the physician must make a prognosis with caution.

The prognosis in a state of atrophy is still more doubtful. Here the physical development and all functions have reached a very low level. The facts that the child is extremely emaciated and in his weight lags considerably behind that of other children of his own age or that his skin is withered and without subcutaneous fat are not decisive; the fate of the child is determined by the heavy losses in his functional capacity which can be regained only after weeks and months, even in the presence of adequate nutrition and even if supplied with breast milk. Again the same three functional disturbances endanger the life of the child: failure to gain weight, intestinal lability and reduction of immunity. The greatest danger comes from the decrease in immunity. Otitis, which regularly accompanies any atrophic condition, so-called para-vertebral pneumonias which almost always originate from atelectatic areas which are secondarily infected, are the most frequent signs of lack of resistance of all tissues. The much quoted phrase by *v.Pfaundler*—"they fall sick due to nutritional damage, they die of infection"—applies particularly to the atrophic child. But even after the atrophy has apparently been cured the reduction in the immunological ability persists for a long time.

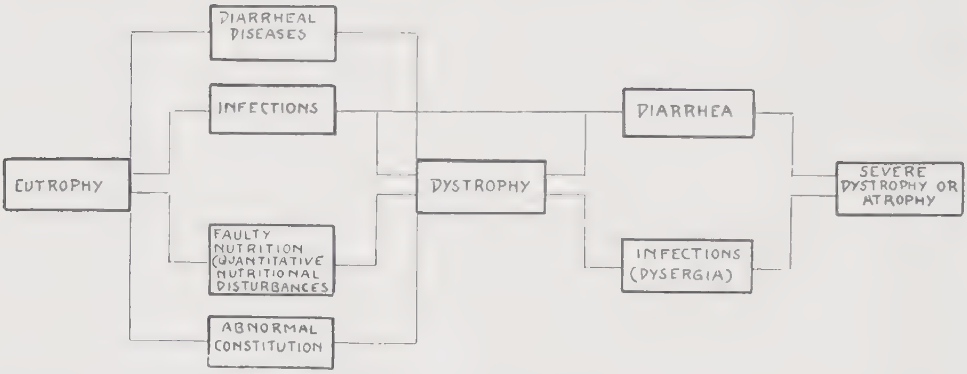
The lack of tolerance of the intestinal tract of the atrophic child has perhaps been overestimated. The over-anxious observation of stools has led to a continuation of quantitative and qualitative under-nutrition for too long a period of time. It was believed that by exercising particular caution in the food intake it was possible to cure diarrheas with their danger to the general condition and to avoid their recurrence. Food tolerance is not a trait characteristic for the body and the individual and is not unalterable. Any diarrhea reduces temporarily the tolerance of the gastrointestinal tract, and this reduction is the more severe the lower the nutritional state of the child. As soon as improvement of the diarrhea starts the lowered tolerance returns to normal as one of the first functions. An infant can still present the picture of atrophy and yet possess again an adequate food tolerance. This observation is of practical significance. The fundamental disturbances in a state of atrophy can never be compensated by a continued inadequate nutrition. A "minimal diet" results in hunger in every single cell and thus lack of some substances which are necessary for normal functions. The atrophic cell can heal only with a nutritional therapy which furnishes the body with all the ingredients necessary for growth and activity. "It is a contradiction to believe that the sick body could be strengthened under the same conditions under which the healthy body would gradually be consumed" (*Finkelstein*). Only the speediest possible introduction of a complete diet can lead to recovery in the dystrophic child. In this connection one will show renewed astonishment at the great tolerance of the intestines with regard to quantity and type of food.

Method and speed of dietetic treatment of dystrophy and atrophy is determined individually on the basis of the factors which caused the deterioration of the nutritional state. While the eutrophic child can be fed with simple milk mixtures which contain additional carbohydrates and perhaps fat (see chap. on Artificial Nutrition), these mixtures are frequently inadequate for the dystrophic child and are completely unusable for the atrophic infant. Alterations of the food become necessary. The stronger tendency to diarrhea must be taken into account. The compensation for the loss of substances which the infant suffered on his way to dystrophy and atrophy and which continues for as long as the child remains in a bad nutritional state, requires a special composition of the diet. In this connection one must take particular care to meet the additional requirements of protein and salts in choosing the diet for a nutritionally damaged child. Breast milk which is poor in salts and protein suffices for the development of the healthy cells but frequently does not, as an exclusive food, fulfill the requirements of damaged cells. The following principles should be considered in the choice of milk mixtures which are used as basis for the nutrition of dystrophic and atrophic children:

Even in the dystrophic infant with normal intestinal activity caution is indicated in the choice of his diet. The tendency to diarrhea requires discretion in any increase of fats and carbohydrates. Acidification of food and feeding mixtures which are relatively rich in protein and salt are preferable to the customary milk-flour-sugar mixtures. The acidification of the food through addition of lactic acid or citric acid already provides more security in this direction. Thus for instance acidified, sweetened whole milk with its high protein and salt content is suitable as a permanent food in the slightly damaged child. An even better protection against recurrence of diarrhea and its consequences in the dystrophic child is sweetened buttermilk, to which roasted flour is added if the intestines function already normally, or protein milk with 5 to 7% sugar.

In the atrophic child breast milk is indicated as a basic food. Only with type-specific food is it possible, particularly in the youngest infants, to rapidly and securely restore the seriously damaged cell functions. The danger of diarrhea is not avoided by giving breast milk. A careful measuring of the food quantities is necessary, at least at the beginning of treatment. A combined diet of breast milk and buttermilk + 5% sugar counterbalances to a certain extent the dangers of breast milk to the intestinal tract of these children. It is safer to start feeding these children at first only with buttermilk and to change to breast milk only when the tendency to diarrhea disappears.

The possibility of a return to a feeding mixture corresponding to the age of the child depends on the degree of damage to the child. In the dystrophic child the attempt can be made after three to five weeks to return to a normal diet. In the atrophic child the special diet probably has to be given for six, eight to 10 weeks. In both cases it is assumed that intestinal activity has in the meantime returned to normal and that the tendency to diarrhea has disappeared.



Fear of intestinal disturbances must, however, never lead to excessive postponement of the necessary complementation of the diet, for restoration and regaining of normal resistance can be attained only with a complete diet. Since every case of dystrophy can, in the last analysis, be traced back to a quantitative or qualitative deficiency of some vital nutritional substances, the correction of this deficiency must be the aim of the treatment. What type of complementation of diet should be carried out in the individual case is determined by the cause of dystrophy through etiologic differentiation (scurvy, damages due to protein deficiency, dystrophy caused by vomiting, etc.). This method of approach also applies to patients who developed dystrophy as a result of an accumulation of infections which also led to a deficiency in vitamins, carbohydrates and protein.

The ways which in the eutrophic infant lead to the state of dystrophy and, in case of prolonged damage and youthfulness of the organism, to the state of atrophy, are presented in the preceding diagram.

The task of the following sections will be to present these causative diseases in detail and to describe their treatment. This includes diseases caused by:

1) Quantitatively or qualitatively inadequate food intake or loss of a smaller or greater part of the food due to structural or functional diseases of the organs serving nutrition.

2) Disturbances in digestion of a suitably composed diet. These are the diarrheas (digestive disturbances).

3) Disturbances in the assimilation of food in the cells with a basically correctly composed diet which is normally digested (Metabolic disturbances).

4) Characteristic for infancy, disturbances of the nutritional state due to infections occurring outside the intestinal tract and primary intestinal infections.

CHAPTER V

Quantitative Nutritional Disturbances

THE DANGERS of inadequate nutrition have been recognized only recently by pediatricians as a cause of disease. As a result of the formerly frequent failures in artificial nutrition of young infants the food was regarded as the factor causing the disturbances in the well being of the children. The apparently logical conclusion from this theory was the belief that by limiting food quantities or individual food components the unfavorable results of artificial nutrition could best be avoided. It thus happened that for many years and decades the dietetic rules during infancy were dominated by the warning against overfeeding. It is certainly justified to warn against the intake of food quantities which essentially exceed the requirements necessary for growth and activity. It is, however, much more dangerous to offer less than the necessary requirements in order to avoid in this manner the supposed dangers of artificial nutrition.

This development in the practice of infant nutrition is hardly understandable today, particularly since already about 50 years ago *Rubner-Heubner* repeatedly emphasized in their presentation of infant nutrition the necessity for an adequate diet. The practical application, however, lagged far behind this observation. For at no time were infants, in the attempt to treat them or prevent diseases, exposed to hunger more frequently than in the period during which the basic laws of energy metabolism were established. Unfortunately this fear of food has not yet entirely disappeared. The recommendation of prolonged "restricted diets," the ordering of rice water for many days, etc., are methods which even today are still frequently applied in the treatment of many infectious diseases or diseases accompanied by diarrhea.

External hunger develops either through insufficient food supply or if part of the food, which is given in adequate quantities, is lost again without being utilized by the digestive processes: for this a qualitatively correct composition of the diet is assumed. If apart from the quantitative deficiency individual substances are lacking (qualitatively deficient nutrition) we find within a short time in addition to general poor development and dystrophy, more or less characteristic symptoms depending on the type of the lacking substance. The significance of the deficiency varies according to the value of the substance which is lacking. Every substance contained in breast milk is essential in the long run and any deficiency or limitation in the supply of one or more of these substances for a longer period of time leads to defective growth and functioning of the organism.

Damage due to external hunger must develop because with any quantitative lack in food intake the organism must fall back on its reserves. In order to maintain body temperature, circulation, respiration, secretions, muscular activity, etc., the reserve substances of the body, particularly glycogen and fat, are utilized in the beginning. The reserves of these substances, especially glycogen, are small in infants with weak musculature and within a short time the organism is forced to draw on vital cell substances (protein, lipoids) the loss of which is connected with destruction of body specific tissue.

This self-consumption of the organism in times of hunger results in a number of symptoms. The children are thin or emaciated, dystrophic or atrophic. The tendency to subnormal temperatures, particularly during the night, is frequently the earliest sign of inadequate food intake. The loss of ability to fight off infections is the most serious symptom developing in the hungry child. And since there is in every case of dystrophy a tendency to diarrhea for even minor causes, a number of these hungry children soon develop diarrhea. These frequent stools are often the cause for incorrect diagnoses and incorrect therapeutic measures. With insufficient food intake the infrequent emptying of stools in small quantities is erroneously expected. However, the starving intestinal cell no longer possesses the ability to normally regulate the bacterial life of the intestines, a defect which favors the development of diarrheal diseases. Hunger is also at times accompanied by vomiting. These symptoms frequently lead to incorrect interpretations, namely, that the child receives too much, and to incorrect therapeutic conclusions. Not every case of vomiting and diarrhea can be explained by an excess of food and it is wrong to treat a condition which developed from a lack of food with a reduction in food quantities.

The danger of hunger also exists in the breast fed child when in cases of inadequate secretion of the breast the addition of other foods is delayed or when the infant is fed exclusively on breast milk beyond the seventh month of life. But even with a most carefully determined artificial nutrition underfeeding is possible if the food intake is calculated on a theoretically correct basis without, however, taking into account the individual needs of the infant. Children of physicians or children whose mothers adhere strictly to one of the many textbooks on infant care, are particularly endangered from that point of view. In institutional care hunger formerly played an important role since its significance in the pathogenesis of many nutritional disturbances was not properly evaluated. The earlier failures which so frequently occurred in infants raised in institutions can today to a large extent be attributed to damages caused by hunger. The infants were fed with too strongly diluted milk mixtures of which children under the wholesale care did not drink enough. There was not enough time to feed the children with the care which is necessary for the feeding of infants. The constant change in nursing personnel also disturbed the eating pleasure of the children. Only after the importance of adequate quantitative and qualitative nutrition was recognized and concentrated feeding mixtures were introduced did the artificial feeding in institutions become successful. The requirement of individual care by one single nurse or two or three steady nurses at the most can even today not be fulfilled in practice.

Whether the food quantity which the child actually took in is adequate can be determined only approximately by a calculation of the caloric value of the food. For the practical application of the calculation it is advisable to estimate the caloric value of the customary feeding mixtures (half milk with 5% sugar, 2/3 milk with 5% sugar, etc.) at not more than 600 calories per liter. Not more than 700 calories per liter should be estimated for concentrated feeding mixtures (whole milk with sugar, etc.) since particularly in mixtures which are rich in fat, large quantities of the ingested fat are not utilized and are lost in the stools and thus withdrawn from the actual nutritional process.

The individual tendencies for developing nutritional disturbances due to quantitative and qualitative hunger vary considerably. Some infants can tolerate even serious faults in nutrition for a long period of time without evidencing damages in their development.

The extensive and important field of nutritional disturbances can best be surveyed by a presentation based on etiological principles. The etiological classification makes it possible to describe characteristic pictures depending on the lack or insufficient supply of one or another or several substances. For the practice of nutrition the establishment of the cause of the disease determines the therapy.

NUTRITIONAL DISTURBANCES DUE TO CONGENITAL OR ACQUIRED DISORDERS OF THE GASTRO-INTESTINAL TRACT

More or less complete obstacles in the path of the digestive organs lead to quantitative hunger.

1) Congenital Stenosis of the Esophagus. Disturbances in the embryonic separation of the Anlagen of trachea and esophagus result in congenital stenosis of the esophagus. The majority of stenoses occur at the level of the biforcation. The length of the affected section varies considerably. The following possibilities exist:

1) Complete absence of the esophagus (rare).
2) Upper and lower segment, both ending blindly, separated by a non-canalized section.
3) One of the segments joins with the trachea or the bronchi:
    a) Upper segment joins.
    b) Lower segment joins (most frequent form).
    c) Both segments join.
    d) Tracheal fistula without stenosis of the esophagus (rare).

The anomaly occurs most frequently in form of a short, blindly ending sack in the uppermost part of the esophagus which is

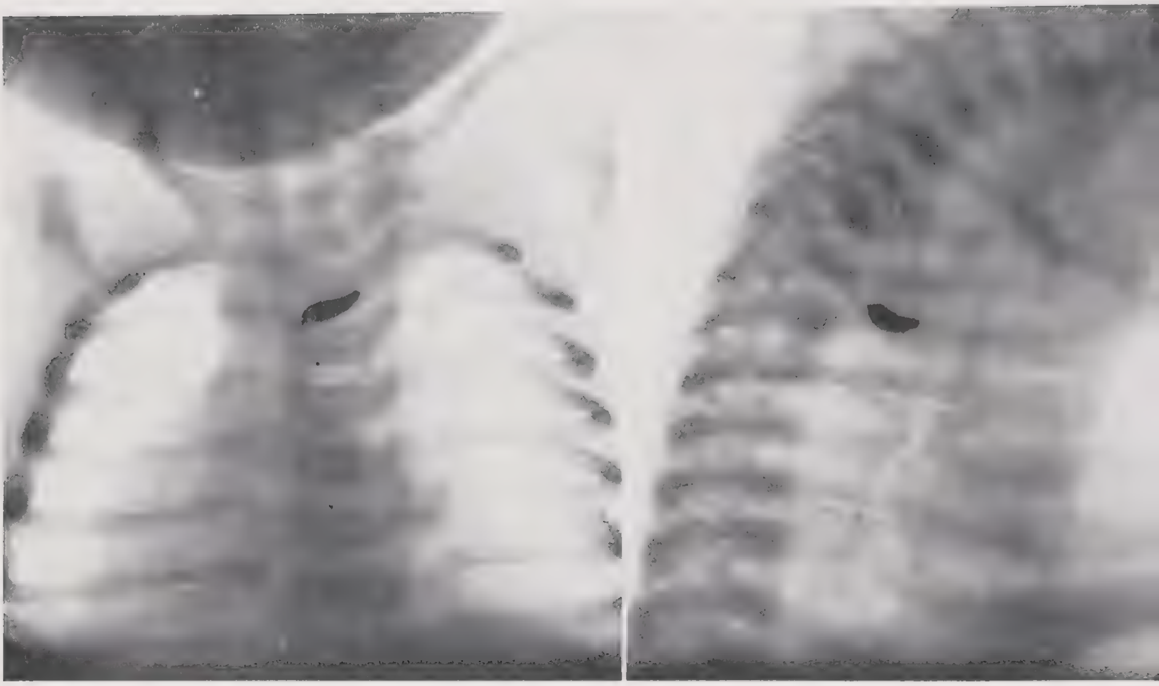


Fig. 21. Infant L.S., two days old. Esophageal atresia. Vomits immediately after first food intake. Lipiodol shows stop in upper third of esophagus. Stomach and intestines contain plenty of air as sign of the existence of a tracheo-esophageal fistula underneath the atresia of the esophagus.

separated from the lower, longer segment of the esophagus by a shorter or longer non-canalized bridge. The communication with cardia and stomach is normally formed. The lower blind sack, occasionally also the part situated above the stenosis, frequently joins with the trachea (see above). This explains why the presence of air in stomach and intestines on roentgen examination does not exclude the diagnosis of esophageal atresia.

Clinical symptoms of the malformation appear a short time after birth. Any intake of liquid is immediately followed by vomiting. The mouth of the infant is filled with mucus which cannot be swallowed. The vomitus is not sour. The milk is not curdled. The introduction of a stomach tube in unsuccessful. A small quantity of lipiodol (barium should be avoided as contrast medium because of a possible communication with the trachea) collects at the end of the blind sack and shows the seat of the malformation.

Until recently all these children died after about eight to 10 days as a result of complete hunger. Through surgical connection of the two blind sacks and closing of the esophago-tracheal fistula it is possible today to keep a small number of these children alive. Earlier occasional attempts to feed the child through a stomach fistula, in the hope of delaying the operation beyond the newborn period, have never been successful.

2) *Brachy-esophagus (Hernia Hiatus Esophagei).* In this probably not so rare developmental disturbance, part of the stomach lies above the diaphragm in the thorax. The pathological symptoms caused by this disturbance appear at times already during early infancy, usually, however, only later when the child receives more solid food or even during adulthood. The children regurgitate or vomit smaller or larger parts of the food during or shortly after the meal. The signs of pylorostenosis are absent (see p. 226). The anomaly can be proven in the roentgen picture where after the intake of barium part of the stomach with its characteristic formation of folds is seen above the diaphragm. If the roentgenogram is taken with the infant's head downward so that the stomach content flows back into the esophagus the section of the stomach which lies above the diaphragm is depicted by the characteristic design of the gastric mucosa. The continuous vomiting which accompanies this disturbance leads to hunger, dystrophy and atrophy. The attempt should be made at first to facilitate the transport of the food in the direction of the pylorus by feeding the infant sitting up and by keeping the child in a half erect position. Cure can be effected by surgical closing of the diaphragm after pushing the section of the stomach back into the abdominal cavity.

3) *Meconium ileus (see cystic degeneration of the pancreas, p. 238).*

4) *Congenital intestinal atresia* constitutes rarely a complete separation of one section of the intestines from the other by a thin septum. More frequently the canalization of a longer or shorter section of the intestines is lacking or the canalization of the intestines is incomplete at one place. This developmental disturbance takes place after the first or second fetal month when the intestinal tract loses the originally established lumen through epithelial proliferation and temporarily turns into a solid cord. If

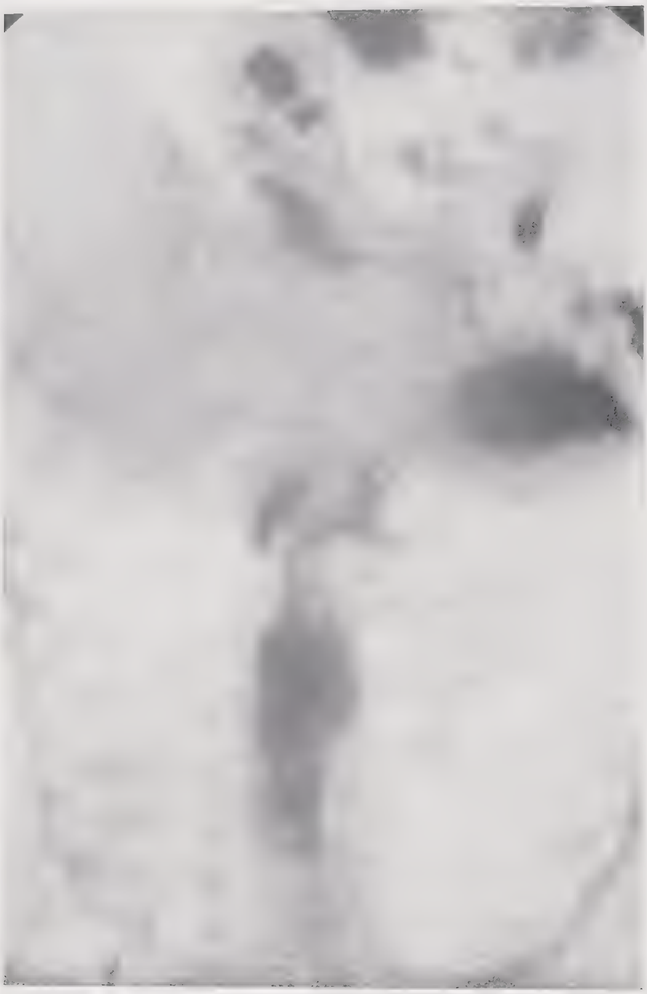


Fig. 22. Infant, M.H., three months old. Hernia hiatus oesophagei. Vomiting few days after birth. Severely dystrophic child. No signs of pyloric stenosis. In the roentgenogram the lower third of the esophagus is above the diaphragm, in the thorax. Contour of the gastric mucosa can be seen in the displaced stomach portion above the diaphragm in upside-down position. Surgery: Closure of the opening in the diaphragm after reposition of the stomach. Very slow recovery.

at a somewhat later period of development the normal second canalization of the cord does not occur, no new intestinal lumen develops. The result is an atresia of the intestines. A longer or shorter section of the intestinal tube remains without lumen. In this manner dividing walls appear in the course of the intestinal tract or a larger or smaller section of the intestines apparently is missing. The intestinal lumen beyond the undeveloped section remains narrow, is, however, always present. This condition is at times, not quite justifiably, diagnosed and described as microcolon. Irrigoscopy proves the presence of the narrow but stretchable section of the intestines. The proof of such an undeveloped colon, containing no gas, must not prevent the decision to surgical correction of the atresia.

Symptoms of an intestinal atresia are violent vomiting from the first day of life. The vomitus also contains bile if the obstruction is below the papilla Vateri. The early beginning of vomiting and the bilious color of the vomitus distinguish this disease from pyloric stenosis. Dehydration and meteorism or even rupture of the excessively stretched blind sack in the course of the intestines develop shortly. If the stenosis is deep-seated no meconium but only small quantities of a greyish-white mass appear from the rectum. If the stenosis is located higher up the intestines can contain meconium which, however, does not include any cornified epithelial cells (which originate from the vernix caseosa and were swallowed by the fetus) (Farber Test*). The meteorism is variable depending on the location of the stenosis: stenoses located in the upper sections usually cause little meteorism, while a low-seated stenosis causes considerable meteorism. In the roentgen picture more or less large parts of the upper abdomen

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* *Farber's test:* Microscopic examination of meconium for cornified epithelial cells and lanugo hair. The intestinal tract, canalized in the Anlage, is closed off from the fifth to tenth fetal week through proliferation of the endothelium. After that time absorption of the proliferated epithelium results in renewed canalization. The fetus swallowed amniotic fluid which in the last two months of pregnancy contains cornified epithelial cells and hair. With normal recanalization hair and epithelial cells are found in the meconium. They are absent in the case of atresia. For examination material from the inner parts of the meconium should be used. The smear is stained with gentian violet for three to five minutes, rinsed with water, decolorized with hydrochloric acid and alcohol. The cornified cells are now well visible.

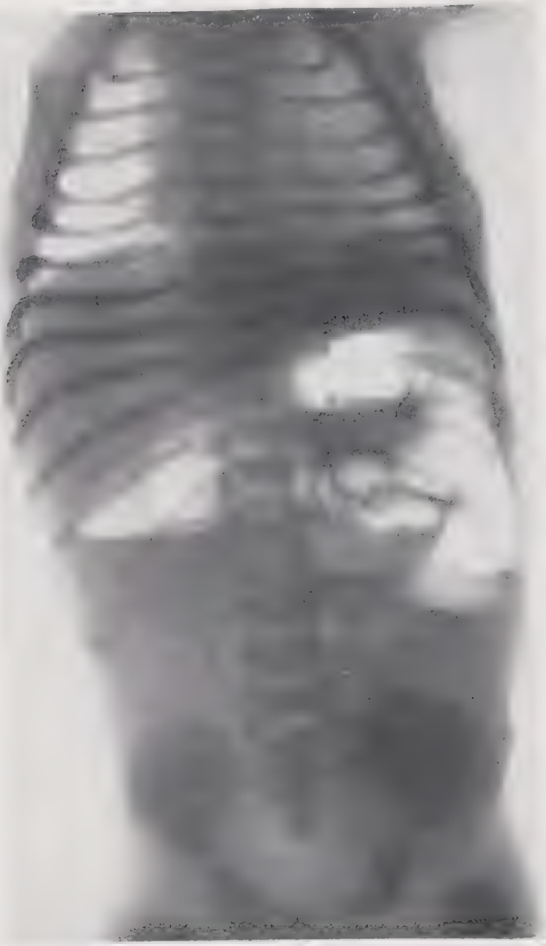

Fig. 23. Infant P.J., two days old. Atresia intestini. P.J. is second child, first child died of intestinal atresia. No meconium. Meteorism. Vomiting. Fluid levels in the upper abdomen, lower abdomen shows no air. Surgery: extensive atresia of the small intestines. Underdeveloped large intestines of the thickness of a thin pencil with a very narrow lumen. Left kidney size of cherry pit with cystic degeneration. Right kidney large.

are seen to contain air. In the intestines one or more fluid levels can be observed. The lower abdomen contains no air. The attempt to present the stenosis through barium intake is not without danger since it is difficult to remove the barium and if vomited the barium can easily be aspirated. Irrigoscopy shows the underdeveloped colon with hardly the thickness of a pencil. Treatment is surgical and should be undertaken as early as possible in form of a side-to-side anastomosis. Because of the great differences in lumen between the closed off upper intestinal section and the undeveloped lower section an end-to-end junction is usually impossible. The prognosis for recovery is not good.

5) *Stenosis of the intestines* occurs when the dividing wall remaining after the second canalization in the intestinal lumen is incomplete, resulting in a more or less narrow connection between the sections of the intestinal tract. Here, too, the section of the intestines above the stenosis is enlarged, the lower section is lacking in development and narrow. If the narrowing of the intestines is considerable there might also be early violent vomiting and a more or less severe meteorism. The symptoms vary depending on the location of the stenosis which can occur from the pylorus down to the beginnings of the large intestines. The child loses weight more or less rapidly. The quantities of stool are small but contain at all times cornified epithelial cells. The intestinal peristalsis is visible. The narrowed sections can be bridged by side-to-side anastomosis. The chances of recovery are better than in patients with complete intestinal atresia.

6) *Malrotation of the midgut* is caused by disturbances in the complicated processes in the course of development of the intestinal tract. Around the sixth to tenth fetal week the rapidly growing intestines do not have enough space in the abdominal cavity (coelom) and crowd into the umbilical cord. As after the tenth fetal week the abdominal cavity grows larger the eventrated part of the intestines returns into the abdominal cavity. The intestines move normally in such a manner that the entire gut from duodenum to the center of the colon (the so-called midgut) settles at first temporarily in the upper left section of the abdomen. By a counterclockwise turn the colon reaches the epigastrum, from here the upper right abdomen, and finally terminates its growth in the region of the right iliac fossa. Now the descending and ascending colon and the mesentery of the small intestines are attached to the posterior abdominal wall. Disturbances in these complicated movements lead to conditions which are referred to as malrotation. In such a case part of the intestinal tract remains

freely suspended and does not become attached to the posterior abdominal wall. Location and extent of the disturbance are determined by the exact time when the normal movements of the midgut are interrupted. Disturbances in the upper intestinal tract can lead to obstruction of the central section of the duodenum and to a volvulus due to lack of fixation of the mesentery. This results in strangulation of the vessels of the mesentery. The slightest degree of non-fixation of the mesentery results in a free cecum. These developmental disturbances occasionally do not show symptoms until after some months or even years of life. Frequently some disturbances and complaints develop already during the first days of life. Bilious vomiting, meteorism, exaggerated and visible peristalsis in the upper abdomen indicate intestinal obstruction. If vomiting continues it comes to rapid collapse and dehydration. The roentgen picture confirms the location of the intestinal obstruction. The treatment is surgical and must include removal not only of the volvulus but also of the obstruction of the duodenum.

7) *"Reduplication" of the intestines.* Scattered epithelial cells from some section of the intestinal tract can develop in close connection with the intestinal Anlage into cyst-like formations; in such cases the endothelium of the cyst does not always correspond to the cells of the intestinal section where the so-called reduplication of the intestines is attached. These formations can occur in any section of the intestinal tract, from esophagus to rectum. Larger reduplications can become obstructions in the intestines. Ileus-like symptoms, vomiting, constipation, pains, collapse indicate the obstruction of the intestinal tract, e.g.: Fig. 16.

Treatment consists in removal of the cystic formations. In young children, as in the previously mentioned patients, it is possible, depending on the location of the disturbance, to decrease the cystic formations by puncture and thus postpone the not inconsiderable operation until a later time.

8) *Invagination or Intussusception.* An obstacle can develop in a hitherto normally functioning and open intestinal tube due to the intussusception and partial fixation of a piece of the intestinal tract in a more distal intestinal section. This can take place already in the first weeks of life. The cause of this phe-

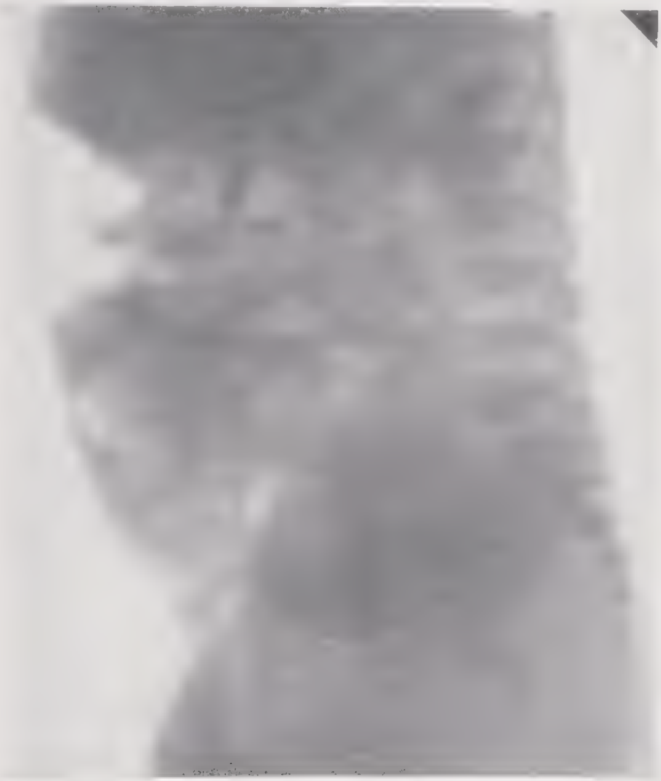

Fig. 24. Infant P.Z. Duplicatio intestini. At birth sacral tumor which was removed. Histologically apparently lymphangioma. At age of one month constipation and anuria. Symptoms resembling ileus. Palpation of a plum sized tumor in the lower abdomen. On rectal examination, the mass is palpated between intestines and bladder. These attacks are repeated a number of times in intervals of several weeks. On filling the cavity with diluted lipiodol: tumor the size of an apple, pushing the rectum to the left on the AP roentgenogram. Loop of the sigmoid pushed upward. Wall of the rectum normal. The cyst has a smooth contour upon filling and reaches almost to the anterior abdominal wall.

nomenon is not known. The organic changes observed in the older child (polyps etc.) are not present during infancy. The disturbance here is functional. Intussusception takes place when the peristalsis in one section of the intestines is more rapid than in the adjoining distal part. This dysharmony in the rhythm of intestinal motions is probably caused by uneven maturation in the vegetative supply of the different intestinal sections.

A hitherto healthy infant begins to cry suddenly and sporadically, as if in pain. The appearance of the child changes. It becomes pale, with rings around the eyes. After some time the child starts vomiting. There are no stools. After the disease persists for some time bloody mucus or blood is discharged from the rectum. Examination reveals a swollen abdomen, painful to pressure. At one place, usually in the upper right abdomen, resistance is encountered. With adequate light protruding intestinal loops or moving peristalsis can be observed even in well nourished children. Palpation reveals a sausage or ball shaped tumor. On auscultation one hears increased bubbling sounds characteristic of obstruction. In the beginning the abdomen appears soft between attacks and only with the onset of painful attacks rigidity and resistance become apparent. The area of the iliac fossa seems at times empty. Rectal examination reveals hypotonia of the sphincter. The rectum is usually empty, but occasionally still filled with remnants of pasty, soft stools. Upon introduction of a thermometer or the little finger traces of blood can be seen on thermometer or finger, however not regularly and not at the beginning of the disease. Irrigoscopy shows an obstruction at some point of the large intestines, since invagination usually takes place through entrance of the ileum into the cecum or through intussusception of part of the colon into adjoining parts of the colon. The picture of the obstruction is typical: depending on the projection it appears on lateral view in form of a semilunar shadow, or in a view in which both intestinal lumina are shown one on top of the other in form of a cockade. In the much rarer occurring invagination of the small intestines (invaginatio iliaca) the discharge of blood in the stool is absent for a long time. Roentgenological presentation of the invagination through irrigoscopy is not always successful in these cases.

Early diagnosis is of decisive significance for the child. Invaginations which are recognized and treated within 24 hours after the onset of the disease have in general a favorable prognosis. Twenty-four to 48 hours after the onset of the disease the prognosis becomes doubtful and only few infants who receive treatment after more than 48 hours survive the major surgical operation which is then necessary, consisting usually of a resection of the part of the intestines which can no longer be separated and which has become incarcerated and necrotic.

In favorable cases, which are not too rare, the invagination reduces itself completely during the transport to the hospital or after irrigoscopy of the patient. If it is not reduced, surgery should be done immediately. The attempt to reduce the invagination by a barium enema given under great pressure and by careful massaging of the intestines is not without danger, particularly if the disease has lasted for some time. However, an attempt should always be made in patients who had fallen sick only very recently. With an early diagnosis reduction of the invagination is successful in many cases. The medication of laxatives or drugs stimulating peristalsis, such as physostigmine and others, is definitely contraindicated. Such a step would only aggravate the invagination.

In preparing for the operation children who already show signs of larger losses of water should receive infusions of saline, glucose or plasma. The child can be fed with small quantities per os already 24 hours after the operation. If the intestines had already suffered considerable damage and the intestinal wall showed hemorrhages and damage parenteral feeding in form of intravenous infusions of plasma, glucose, amigen with the addition of vitamins (especially vitamin C) should be continued for some time (two to four days). All these children have high temperatures following the operation. They are the result of always present irritation or inflammation of the peritoneum and damages of the intestinal wall. The incipient peritonitis can usually be overcome by giving antibiotics ($^1/_2$ g streptomycin plus 6 x 50,000 units penicillin daily). Since it is possible to feed children for many days parenterally and to cure the secondary infection, the prognosis in the treatment of invagination has improved considerably, even in patients where surgical intervention was delayed. Errors in diagnosis occur when appendicitis (very rare during infancy) or, because of bloody stools, bacterial dysentery is erroneously diagnosed.

REGURGITATION AND VOMITING

One of the most frequent causes for the development of hunger conditions are losses of food through regurgitation and vomiting. Vomiting occurs much more frequently during infancy than at any other later period of life. At times the cause for the vomiting can be determined. Infections, frequently of a minor nature, a cold, a catarrh suffice in a large number of children to stimulate the stomach to abnormal elimination. Some children vomit only one single time; this occurs suddenly and is not infrequently the earliest sign of a beginning infectious disease. In other patients there is continuous vomiting throughout the course of the disease. Vomiting is also frequently an early and important symptom of acute nutritional disturbances.

In the majority of children who vomit, however, none of the above causes can be found. This applies particularly to infants who for a longer or shorter period of time regurgitate part of their food after almost every meal. This has been referred to as habitual vomiting. The quantities of food which are regurgitated vary greatly. The time after the meal at which the vomiting occurs also varies.

All children suffering from so-called habitual vomiting should, for diagnostic purposes, be examined roentgenologically. In a large number of patients a roentgen examination of the stomach reveals anatomical and functional peculiarities which explain the vomiting and regurgitating. and thus permits the application of suitable therapy. Apart from the uncommon gross anatomical changes such as short esophagus, hernia through the hiatus oesophagei (see p. 207), diaphragmatic hernias, malrotation of the midgut (see p. 211) and others, we find the following more commonly occurring abnormalities: partial volvulus of the stomach, a "cascade" type of stomach caused by a strongly dilated transverse colon, a so-called chalasia oesophagei consisting of a weakening and dilatation of the lower part of the esophagus which permits a reflux of part of the gastric contents, and others more. By placing the child in a position which compensates for the abnormal position of the stomach it is possible to prevent vomiting and regurgitating and to permit normal development of the child: e.g., keeping the child in a constant sitting position in case of chalasia oesophagei; placing the child in prone position and feeding it in this position in case of partial volvulus of the stomach; placing the child on the abdomen, with the upper part of the body raised, in case of a "cascade" stomach which is caused by pressure of the colon. On the basis of observations made on fluoroscopic examination, after feeding of contrast medium, it is possible to adjust the mechanical irregularities by suitable placement and feeding of the patient. The more carefully the anatomy and function of the gastro-intestinal tract is examined and analyzed in these patients, the easier it will be to control the occurrence of so-called habitual vomiting.

It is not always possible to find an explanation for the vomiting. Vomiting might be induced partly through mechanical factors (inadequate feeding technique). Children who drink awkwardly and hastily or who cannot grasp the nipple airtight during sucking due to a strongly receding chin swallow with the food large quantities of air which collect in form of an air bubble in the upper part of the stomach. If the air leaves the stomach through the cardia, with the child lying down, it carries along part of the food still in the stomach. The old custom to have the children eructate after drinking is therefore justified. In many children this is sufficient to cure minor cases of vomiting. Placing the child in a half-sitting position has a similar effect. In other young infants the motility of the stomach seems still incompletely developed. A reflex, caused by a stimulus of the ingested food upon the gastric wall, is lacking. This reflex should bring about a contraction of the gastric wall which tightens its grasp on the food contained in the stomach. The absence of this so-called "peristolic reflex" (the existence of which has been questioned) favors the development of vomiting. This delayed development of the peristolic reflex results in a condition which resembles that of an atonic stomach.

Disturbances of the secretory functions of the stomach have not been proven in infants suffering from vomiting. A certain amount of hyperacidity is occasionally encountered, more frequently, however, it is absent. The secretion of the individual enzymes is always undisturbed. The assumption of a "hyper-sensitivity" of the gastric mucosa has not been proven.

It is no doubt wrong to search for the cause of habitual vomiting only in the stomach. At times—and this has been corroborated by certain therapeutic successes—the causes might be disturbances of the nerve supply of the stomach (vegetative system) which interfere with the motility of the stomach. In other children irregularities in the activity of the abdominal muscles and the diaphragm might be responsible for the development of vomiting. The musculature of the abdominal wall and the diaphragm are always essentially involved in the act of vomiting. The strong agitation of the abdominal musculature has repeatedly been observed in the vomiting child so that one is almost tempted to speak of "grimacing of the abdominal musculature" in these children.

It is not possible today to indicate in every patient the etiology of habitual vomiting. On the basis of practical experience it can, however, be assumed with certainty that there are different types and forms of vomiting. The quantities of regurgitated food, first of all, show great variations. By weighing it can be confirmed that even in the simple regurgitation following meals up to 50% or more of the ingested food is again lost. If regurgitation is increased to vomiting it happens not infrequently that the entire meal, possibly also with the addition of mucus and gastric juice, is regurgitated. The act of vomiting itself can take one of two different forms: one resembles the processes of vomiting as observed in older children and adults. With a few retching motions and exertions of the abdominal muscles the food is ejected in a stream. In other children smaller portions are ejected spasmodically immediately following a meal. The absence of nausea which precedes the vomiting is remarkable and characteristic, in contrast to adults and older children. The infant occasionally looks somewhat pale, the eyes appear wide open and shining. But after the vomiting the children are immediately cheerful again and ready for eating. A short anxious crying might follow the vomiting. This form of habitual vomiting whereby the food is obviously ejected with a certain amount of muscle tension, has been referred to as "spastic vomiting," in contrast to the so-called "atonic vomiting." In this case the gastric contents flow slowly in a thin stream, intermittently or continuously, from the corner of the mouth. There seems to be no discomfort. Furthermore, there are no stronger muscle tensions, the children lie awake, quiet and content. Usually slight chewing motions and movements of the tongue can be observed. This atonic vomiting resembles to a certain extent the rumination of animals, a process which also appears not too infrequently in the infant (see p. 223).

Habitual vomiting hardly ever exists from the time of birth. It develops in the second and third weeks of life, although it has been impossible to find any external damage as causative factor. It persists as the only symptom for a number of months and disappears around the fourth or fifth month of life. It seems to occur more frequently in breast fed infants than in bottle fed infants. Furthermore, a diagnosis of habitual vomiting might more readily be made in the naturally fed child than in the artificially fed infant where there might be a suspicion that the vomiting is a result of errors in the quality or composition of the diet. The general condition of young regurgitating children need not be disturbed. The influence upon body weight is also insignificant, provided the ejected quantities are small. Vomiting might persist for weeks without any weight losses. Some children even continue to gain in weight.

Vomiting can lead to an interruption in development only when the amounts of regurgitated food are so large that the requirements of the child are no longer met. The ejection of small quantities of food need not disturb the development of the children. In practice it is without significance. It is like a blemish in the nutritional process, which does not require correction unless the anxious relatives overestimate the symptom, draw the wrong conclusions and attempt to cure the ailment by radical measures such as decrease in food quantities, stronger dilution of food, etc. In most patients it is certainly an error to trace back all habitual vomiting to over-feeding, incompatibility of ingested food, unsuitable composition of breast milk or an "upset" or "weak" stomach.

Vomiting in the older infant should be evaluated altogether differently. If in a child beyond the third month of life, whose gastric functions had been undisturbed until then, sudden severe vomiting sets in, the cause will in all probability be external damage. This applies particularly to the older breast fed infant where the occurrence of vomiting is almost always the first sign of some parenteral infection. This central toxic vomiting frequently also affects the general conditions. Even if this type of vomiting occurs only once it suffices in many children to bring about a considerable weight loss of several hundred grams which are rapidly regained if vomiting is not repeated and if the infection does not cause an acute diarrhea (see p. 459).

Infants suffering from habitual vomiting usually belong to a certain type. In *Sigaud's* classification of different types of constitution the majority belongs to the typus cerebralis (see page 488). They are usually skinny children with thin bones whose adipose tissues develop only slowly. Frequently they are restless, nervous children who do not sleep much. The bristly hair which often grows very profusely extends far down on the forehead. The hair on temples and in front of the ears also persists for a long time in these vomiting children.

Since is it impossible to determine in the individual case the cause of habitual vomiting, treatment becomes more difficult. It is therefore indicated to try out the empirically tested measures with regard to nursing care, diet and drug therapy, simultaneously or one after the other until a method can be found which in the individual case causes the vomiting to disappear. Measures regarding the care of the child should begin with nursing. Swallowing of air during sucking on the breast or from the bottle should be avoided as far as possible. If the child is nursed on the breast care must be taken that the child's lips grasp not only the nipple but also the areola. Breathing through the nose must be maintained by keeping the breast away from the nose. In some cases it is advisable to place the child, not as usual lying down on the mother's lap but rather in a position recommended by French authors whereby the child is held vertically in front of the mother during nursing. In bottle fed infants too large a hole in the nipple encourages the swallowing of air. Care must also be taken that the milk fills the nipple at all times during the meal. This can be accomplished by a gradual raising of the bottle during the meal. By feeding the infant in a prone position, with the child's chest resting on a hard pillow and the head extending beyond the pillow, it is possible to reduce the swallowing of air and thus improve some cases of habitual vomiting. As mentioned previously, however, it should never be forgotten to let the child expel air in upright position during and after the meal in order to remove the swallowed air without the loss of food.

Dietetic treatment must above all be free from the assumption that habitual vomiting is due to excessive food intake. Children who vomit usually receive through this loss of food too little rather than too much. Malnutrition can cause vomiting, as has been mentioned previously. Stronger dilution of feeding mixtures or reduction in food quantities therefore never bring about an improvement unless overfeeding on the breast or the bottle caused the vomiting. This can be determined by weighing the food intake. By shortening the drinking time on the breast or by reducing the food quantities in artificially fed infants, overfeeding and thus the cause for the vomiting can be removed. Stimulation of the supposedly incompletely developed peristolic reflex brought progress in dietetic management. This can be accomplished by adding four to five teaspoons of a thick cereal prepared with whole milk (farina), given before the meal, while the remaining food (breast milk or artificial food) remains unchanged. The added cereal must be taken into account in calculating the total food quantities. In many patients the addition of cereal before the regular meal is not sufficient to improve vomiting. In some patients vomiting will cease only if the entire food is given in concentrated form, in others again more frequent (six to eight) and smaller meals will bring about an improvement. The so-called "double food" is suitable for concentrated feeding mixtures, such as whole milk with 17% sugar, mixtures of which about two thirds or one half of the quantities given in ordinary milk mixtures suffice to cover the nutritional requirements. If small quantities of these concentrated food mixtures are ejected, the quantity remaining in the stomach is still sufficient for the development of the child. Particular care must be taken to cover the fluid requirements in cases of exclusive feeding of any concentrated and usually also protein-rich diet, especially on hot days and in hot climates. This can be accomplished by frequently offering small quantities of tea, sweetened with saccharine, between but not shortly before the regular meals. If this method of feeding results in a weight gain, even a continuation of vomiting can be accepted without worrying, since these gastric symptoms disappear in almost every case during the fourth month of life at the latest.

At times it is also possible to cure vomiting by changes in the colloidal composition of the food. This can be done by increasing the concentration of rice water (10% instead of the customary 2 to 3% rice water, which, however, must be made of whole rice and not rice flour) with the addition of 2 to 5% gelatine or the addition of substances prepared from carob flour which brings about considerably greater changes in colloidal composition (Nestragel). One to 2% of these substances are added to the milk mixture.

A great number of drugs have been recommended for treating habitual vomiting. They are supposed to partially correct the assumed imbalance in the vegetative system or have a centrally depressing effect. Here, too, it is not possible to judge from the vomiting or the behaviour of the child which group of drugs would be indicated in the individual patient. Patients in whom all drug therapy fails are by no means rare.

Atropine or eumydrine and papaverine act on the vegetative system. The infant receives atropine and eumydrine in a solution of 0.01:10.0g, two drops before every meal which can be carefully increased to four to five drops if the smaller quantities have no effect. The appearance of an atropine rash does no harm but indicates that the limit in the quantity of this drug has been reached. Papaverine which reduces the increased tonus of the smooth musculature seems to have less effect if given internally (0.005 to 0.01g per dosis) than given subcutaneously (once a day 0.01 to 0.02g). A mixture of papaverine hydrochloricum 0.01, Pyramidon 0.05 per os twice in 24 hours has also been recommended. A centrally depressing action upon the vomiting center can also be obtained by phenobarbiturates (Luminal 0.03–0.045, 2–3 times daily) or 1/4 to 1/3 tablet of a brom-urea preparation (e.g., Adalin). As a result of this, the children remain continuously in a light sleep (see pylorospasm).

In the older infant who vomits several times at the beginning or in the course of an acute infection, one stomach lavage frequently stops the vomiting. For this the complete filling of the stomach with slightly alkaline solution is more important than complete emptying of all food remnants and mucous substances. After the lavage a larger quantity of liquid (100 to 150g) should be left in the stomach.

RUMINATION

Rumination is a form of vomiting peculiar to the older infant, which in its nature is one of the "habits" of the child. Psychological causes contribute to the development of this unusual form of vomiting. This assumption is supported by the fact that rumination disappears in the course of the second year when the sphere of interest of the child is enlarged. Spontaneously or following a disease which was accompanied by vomiting, sometimes cases who were hospitalized or ill for a long period of time, children between the ages of six to 12 months begin to regurgitate into the mouth smaller or larger quantities of food. After some chewing motions the greater part of the regurgitated food is again swallowed. Some parts of the food are lost in this process; partly it flows from the mouth or, if the child ruminates in prone or sitting position, it falls down on the sheet, which happens not infrequently.

The mechanisms whereby the child regurgitates or ruminates the food vary: either the children place several fingers or the fist into the mouth and thereby produce a gagging and vomiting reflex, or other children manage somehow to pump the food back from the stomach into the oral cavity by means of the tongue. Chewing and swallowing of this sour food apparently does not produce an unpleasant sensation for the children. On the contrary, one gains the impression, judging from the facial expressions, that ruminating is connected with feelings of satisfaction. The sheets of children who can already crawl in bed have such a typical appearance that this alone permits a diagnosis. Scattered over the entire bed and over the clothes are wet and dry spots produced by the spasmodically and constantly lost food. These losses are frequently so great that the development of the children suffers. The results are interruptions in weight gain lasting for weeks and months. There are no organic changes in these children. In the roentgen picture one sees the opening of the cardia and relaxation of the esophagus during the act of rumination. It is possible that the diaphragm also participates in the regurgitating of the gastric content. Treatment of this pleasurable habit is difficult. Thickened food which becomes liquid in the stomach does not cure or improve the condition. Addition of colloidal substances which make the gastric content more solid and more viscid, such as substances prepared from the pods of carob (e.g., Nestragel) seem to be effective in light forms and by early institution of treatment. Tying the hands or putting on cuffs which make it impossible for the child to place the finger in the mouth, help only rarely since the children find other methods to produce the pleasant ruminating of sour food.* If it is possible to distract older infants with toys or some intensive activity, ruminating sometimes stops and the child finds satisfaction in some other activity. Closing of the esophagus by introducing an inflated rubber finger into it which has been recommended for serious cases should be reserved for the most extreme forms only where there is danger that the child might be severely damaged in its nutritional state by hunger. Even obstinate cases of rumination are gradually cured in the second year of life when the child is distracted from his ego and gains new interests in play and speech.

PYLOROSPASM AND PYLORIC STENOSIS

If an infant of about three to six weeks of age develops vomiting of increasing severity and in a particularly violent form so that with regularity following every meal larger food quantities are ejected in a projectile manner, anatomic changes must be assumed in these patients such as hypertrophy or spasm of the pylorus with more or less complete obstruction.

The newborn occasionally shows symptoms during the first days of life which are very similar to those encountered in pylorospasm. Newborns during the second to tenth day of life begin to vomit all their food. There is visible peristalsis, a pyloric tumor can be felt. This condition disappears in the newborn after three to four days. Treatment is not necessary. Stenotic conditions in the small intestines which lead to partial obstruction can present clinical manifestations which resemble pylorospasm. If the stenosis is located below Vater's papilla—and this is mostly the case—the vomitus contains bile. The vomitus of the pylorospastic child is always sour and consists of gastric content, that is, casein curds and gastric juices.

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* At times a change in taste of the stomach content is useful, e.g., by chloromycetin.

Pseudo-pyloric stenosis of the newborn has also been brought in connection with a hypertrophy and dysfunction of the adrenal cortex (Piries Syndrome). Cortisone and salt are assumed to terminate vomiting in severe cases.

In the very first days it is not always possible to find definite symptoms of the disease, the so-called pyloric stenosis. After three, four and up to eight days positive symptoms appear. Vomiting comes rapidly and as a surprise, as there is no preceding nausea. Only in a few children the vomiting is preceeded by anxious, painful and brief crying. Vomiting is explosive. Already during or at the end of the meal, but even 15 to 30 minutes later, larger or smaller parts of the meal, also remnants of previous meals, are ejected with force. A diagnosis of pyloric stenosis is certain when peristaltic waves of gastric motions become visible. But here, too, errors are possible. Peristaltic waves are by no means rare in young children with active peristalsis and thin abdominal walls. Before any vomiting, also from different causes, the stomach stiffens and becomes visible under the thin abdominal walls in form of a ball-shaped swelling. The peristaltic wave in pyloric stenosis always begins under the left rib bow. It slowly moves from left to right across the middle line or even further

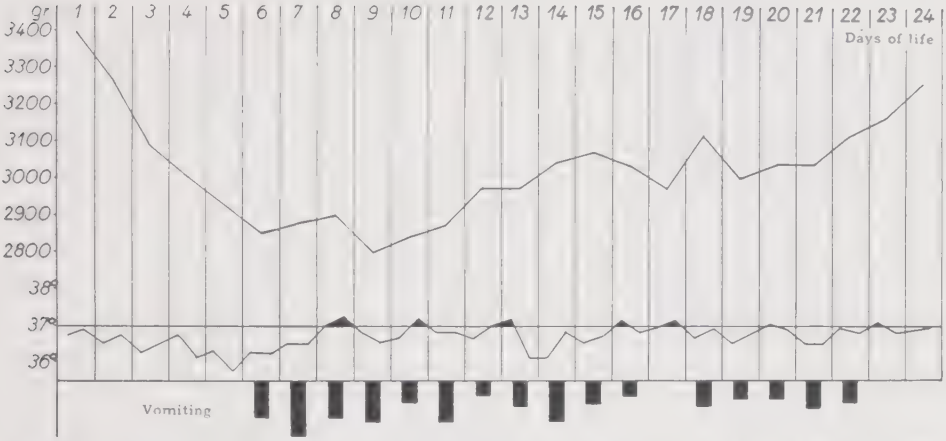

Fig. 25. Infant L.T. Projectile vomiting from the sixth day of life, visible peristalsis of the stomach, delayed gastric emptying on roentgenography. Spontaneous cessation of vomiting at the end of the third week of life. Transitional pylorospasm of the newborn.

toward the axillar line. Anti-peristalsis is never observed. The path of the visible wave does not change, as it does in visible, active intestinal peristalsis. It requires patience to observe this symptom. It can best be accomplished if the child drinks small quantities of food and if the stomach is then observed carefully for several minutes with good lighting from the side. Stroking of the abdominal wall and stimulation with cold application cause active peristalsis. A few days after the onset of vomiting it is possible in most patients to palpate as a further symptom the thickened, spastic pylorus to the right of the middle line, as a mass the thickness of a pencil, the length of a joint of the little finger, hard and elongated. At times it can clearly be felt how the pylorus hardens from time to time, under the approaching wave, and then again relaxes and can no longer be palpated.

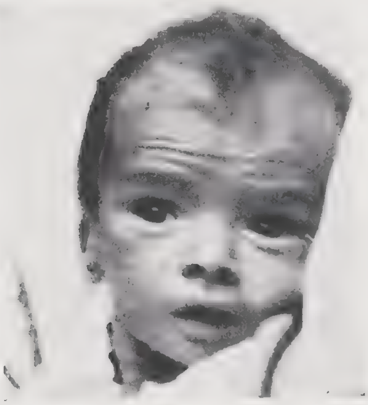
Fig. 26. Wrinkling of the forehead, dense hair growth in a child with pyloric stenosis.

Pyloric stenosis occurs only partly in children of nervous parents and in children who show neuropathic signs. Many of these infants belong to the typus cerebralis. It has been repeatedly observed that several children in one family suffer from pyloric stenosis. It occurs more frequently in boys than in girls. According to some statistics there are 80% boys and only 20% girls. Many of the children are thin, with a dense dark hair growth. The head hair reaches far down on the forehead, the eyebrows frequently are strikingly thick. Most pylorospastic children have wrinkled sinister looking foreheads (see Fig. 26).

As a result of the considerable food losses due to severe vomiting the children develop a number of further pathological symptoms. The abdominal walls sink in. If only small amounts of food pass through the pylorus which is closed off by spasms, there is no stool formation. In the severest forms of the disease real, dark hunger stools are eliminated. On the other hand there are also children where even in pylorospasm hunger produces diarrhea, whereby small quantities of mucous stools are eliminated several times a day. Urine secretion is reduced and the urine, as a sign of dehydration, contains leukocytes and traces of protein. The cause of the temperature elevation which occasionally occurs in the pylorospastic child cannot yet be fully explained. The spastic closure of the pylorus produces a considerable delay in the emptying of the stomach. Not only do the individual meals remain in the stomach for many hours, but as the disease progresses we find ptosis, or rarer dilatation of the stomach, and food remnants remain there for many days and begin to decompose. Secretion of gastric enzymes and hydrochloric acid is undisturbed, frequently even increased. The hydrochloric acid which is emptied into the stomach is completely lost to the organism due to the vomiting. The quantities of acid ions in the organism are thus reduced, resulting in a compensated, occasionally even uncompensated alkalosis.

Roentgen examination of the stomach reveals some remnants of fluids even after a longer pause in food intake. After ingestion of barium large amounts of remnants can be found even after two hours. Further symptoms, apart from the occasionally absolute delay in transition of barium from stomach into duodenum, are: 1) hyper-peristalsis of the stomach; 2) hypertrophy of the gastric wall, and 3) dilatation of the stomach, if the disease persists for a longer period of time. This permits a certain amount of differentiation in the roentgenologic examination with symptoms encountered in habitual vomiting, where: 1) the stomach is never dilated; 2) emptying of the stomach is not delayed at all or only delayed very briefly, and 3) the intestinal passage frequently is particularly rapid. According to the length of stay in the stomach, downward dislocation of the stomach, filling of the stomach with air without corresponding filling with air of adjoining parts of the intestines, certain conclusions can be drawn for judging the severity of the individual case and the method of therapy.

The constitution of the child seems of importance for the course of the disease. Two types can be differentiated: part of the infants react to a similar kind of violent vomiting already in the first days of the disease with loss of weight, general weakness and development of signs of dehydration. A second group of patients shows only moderate or very gradual reduction in weight and develops only a light or moderate case of dystrophy. In hydrolabile children which are threatened, with the onset of pylorospasm, by a rapid development of dehydration and dystrophy, any further day of hunger represents a most serious danger. Hydrostabile individuals who only gradually develop signs of dystrophy can much better tolerate hunger caused by vomiting. If pylorospastic children who develop dystrophy within a few days also show signs of dehydration, the condition becomes serious. These severe symptoms hardly ever develop in children who can maintain their weight for a longer period of time despite vomiting.

In contrast to habitual vomiting there are in pyloric stenosis anatomical changes of the pylorus, which shows a tumor-like thickening. This pyloric tumor originates from a real hypertrophy primarily of the ring muscles and less of the longitudinal muscles of the pylorus with a thickening of the individual muscle fibres. The thickening of the musculature continues in the duodenum and stomach in the parts adjoining the pylorus. It can extend as far as the fundus and the esophagus where the musculature then assumes a strikingly white and tough appearance. The strong wrinkling of the mucous membranes which, if the disease persists for a longer period of time, becomes slightly thickened, even altered in the sense of a gastritis and superficially covered with erosions, contributes actively to the narrowing of the pylorus. In some patients the unusual shortening of the stomach mesentery and of the suspensory ligaments of the stomach is of significance for the development of passage disturbances; these fix the pylorus tightly in a somewhat kinked position.

The development of muscle hypertrophy of the pylorus has not yet been explained. The assumption that it is a secondary hypertrophy due to overexertion in the initially only spastically contracted muscle must at present be abandoned in favor of the theory of a primary hypertrophy since it has been possible to prove hypertrophy of the pylorus even in fetuses and newborns. Hypertrophy of the pylorus is accompanied, under the influence of as yet unknown stimuli, by a spasm of the hypertrophic pylorus. Hypertrophy and spasms then lead to the development of the symptoms of pyloric stenosis. Perhaps lack of balance of the vegetative system in the nature of an increased tonus of the vagus is responsible for the development of the symptoms. The fact that spastic symptoms disappear after the third month of life despite continuation of hypertrophy of the pylorus speaks for the action of temporary nervous stimuli.

> Other authors have assumed that pyloric stenosis and pylorospasm belong to the hormonally conditioned pregnancy reactions of newborns. With the absence of defense substances or the inability to produce them, the hormones which are transmitted from the mother to the fetus produce a hypertrophy of the smooth musculature which in girls leads to actually proven temporary hypertrophy of the uterus. In boys these hormones act upon the smooth musculature of the pylorus. This interesting hypothesis would explain the more frequent occurrence of pyloric stenosis in male infants. This theory can explain the development of hypertrophy even before birth, but not the occurrence of spasms in the musculature.

Findings have also suggested that the development of the plexus myentericus is incomplete in the pylorus, similar to Hirschsprung's disease (see p. 471) whereby the defect lies in the lowest section of the intestines. The disturbance in the balance of the vegetative innervation leads to spastic conditions in this section of the intestines and secondarily to hypertrophy. The success of surgical treatment (see below) shows that the severance of sympathetic nerve fibres is perhaps more important for curing the disease than the cutting of the musculature. The spasms of the musculature are in any case more important for producing symptoms of the disease than the tumor-like thickening of the muscle. This is proven by the fact that muscle thickening can exist for two to three weeks or more before the onset of the disease without causing symptoms and that the disease can be cured although hypertrophy of the pylorus persists for months, possibly even years.

The closure of the pylorus, which originates in this or another as yet unknown manner, is very tight. Water pressure of 100 cc is required in the stomach of a child who had died of pyloric stenosis, in order to force the pylorus open, while a normal stomach, even the contracted stomach of a cadaver, which can resemble the stomach of a pylorospastic child, unfolds already under a water pressure of 30 cc.

In the treatment of pyloric stenosis it is impossible to recommend routinely one method or another. Medicinal as well as surgical therapy are justified. They must be adapted to the condition of the individual patient. An attempt should always be made at first to treat the patient medically. If medical treatment, however, does not show a rapid success in form of weight gain of the child, surgical treatment is indicated. Surgery must not be delayed too long to avoid operating the child in a poor nutritional state. Prolonged medical treatment can therefore be applied only to hydrostabile patients who show at least a slight weight gain despite vomiting and hunger under dietetic and drug therapy. Pylorospastic children who are hydrolabile, who show rapid and considerable weight losses or signs of dehydration, should immediately be operated upon. Social circumstances also influence the indication for surgery. If it is impossible to carry out treatment lasting for several months in the home or in the

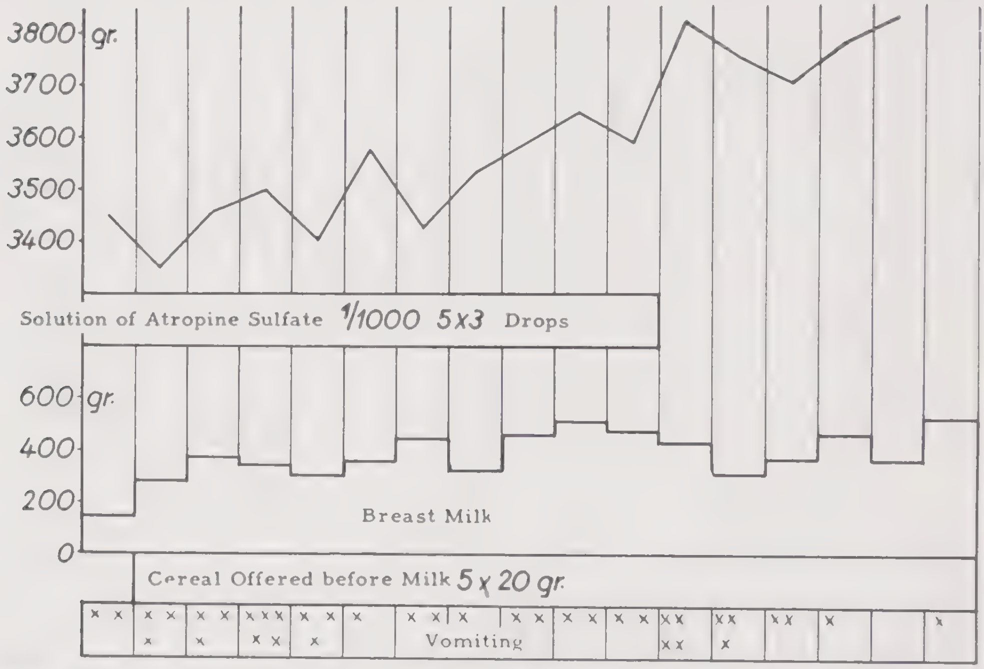

Fig. 27. Infant G.A., four weeks old. Typical picture of pyloric stenosis. Vomiting, visible peristalsis, delayed emptying of the stomach. Cure effected with atropine and thickened cereal feeding before meals.

hospital, it is advisable to cure the disease in a few days through the relatively safe operation.

With medical therapy dietetic treatment is of prime importance. The difficult problem to be solved is, how to reduce vomiting and at the same time avoid hunger. The choice of diet is of less importance. Pyloric stenosis can be cured with any type of diet. Complete feeding mixtures are required also in these cases. The earlier recommended low caloric, strongly diluted feeding mixtures containing little or no fat are not advisable. The food can be given in 5–6 larger meals or in more frequent meals (eight to 10 daily). The quantity of food whereby the child does not vomit any more, must be determined cautiously. Here, too, it is not advisable to adhere to a certain procedure. In one case the method of fewer and larger meals might be more successful, while another patient will fare better with frequent small meals.

A certain progress in the medical treatment of pylorospasm was seen in the partial or complete feeding with concentrated liquid or semisolid feeding mixtures. This includes the pre-feeding of cereal in the breast fed as well as the artificially fed infant. In the breast fed child two, three, or four teaspoons of a thick cereal are given every time before the child is put on the breast; this cereal is prepared from 100g expressed breast milk, one to two teaspoons fine farina and one teaspoon sugar. If the mother is unable to express milk manually the cereal can be prepared from whole milk, farina and sugar, as in the artificially fed child. If this procedure is not sufficient, an attempt must be made even in the breast fed child to give the entire food quantity in concentrated or solid form. The necessary quantity of food is supplied in form of cereal prepared with farina which can be made richer in calories by the addition of powdered milk. Fat should not be added in order to avoid prolonged retention and decomposition in the stomach. With any kind of concentrated feeding particular care must be taken to meet the fluid requirements of the infant. An attempt can be made to give the child water in smallest quantities between the meals. If this produces nausea the water requirements must be met by rectal instillations or parenteral infusions.

Many types of medicines have been recommended for drug therapy of pyloric stenosis. The value of all these drugs is questionable, at least not predictable with any regularity and certainty. Under the assumption that the spasm of the pylorus is caused by an increased tonus of the vagus a number of drugs has been recommended, such as atropine, eumydrine, papaverine in combination with pyramidon and adrenalin. Regular successes can be expected here just as little as in functional vomiting of the infant. The occasional use of narcotics seems to be preferable, particularly in children where spasms of the pylorus are connected with marked discomfort, restlessness or pain. In such cases vomiting can better be reduced by narcotics (luminal three times daily 0.015–0.03, Adalin two to three times 0.15 to 0.25); this keeps the children constantly in a light sleep.

Gastric lavage will have no curative effect. Frequent lavages are harmful, as they favor the development of a dilatation of the stomach.

If a child suffering from pyloric stenosis fails to gain weight after two to five days despite dietetic or drug therapy, or if the general condition of the patient deteriorates rapidly or if there is a social indication which prevents medical treatment, which is always of long duration and never simple, then surgical treatment is indicated. The operative successes with the now customary *Weber-Ramstedt* method are good in the hands of an experienced surgeon. Provided the technique is mastered and the dangers of the operation are known, the mortality is low. However, no less important than the skilled carrying out of the operation is the correct post-operative treatment of the patient. Only a close cooperation between surgeon and pediatrician can ensure success of the operation.

The operation must include adequate preparation of the patient. In children whose nutritional state has already more or less deteriorated, who had lost weight and are withered and dehydrated, the water supply is replenished shortly before the operation through intravenous or intratibial infusion of saline, glucose and plasma. Before the operation the child receives three drops of an atropine solution ($^1/_{1000}$). A catheter is introduced through the nose into the stomach in order to remove food remnants and air from the stomach. The catheter remains in place during the

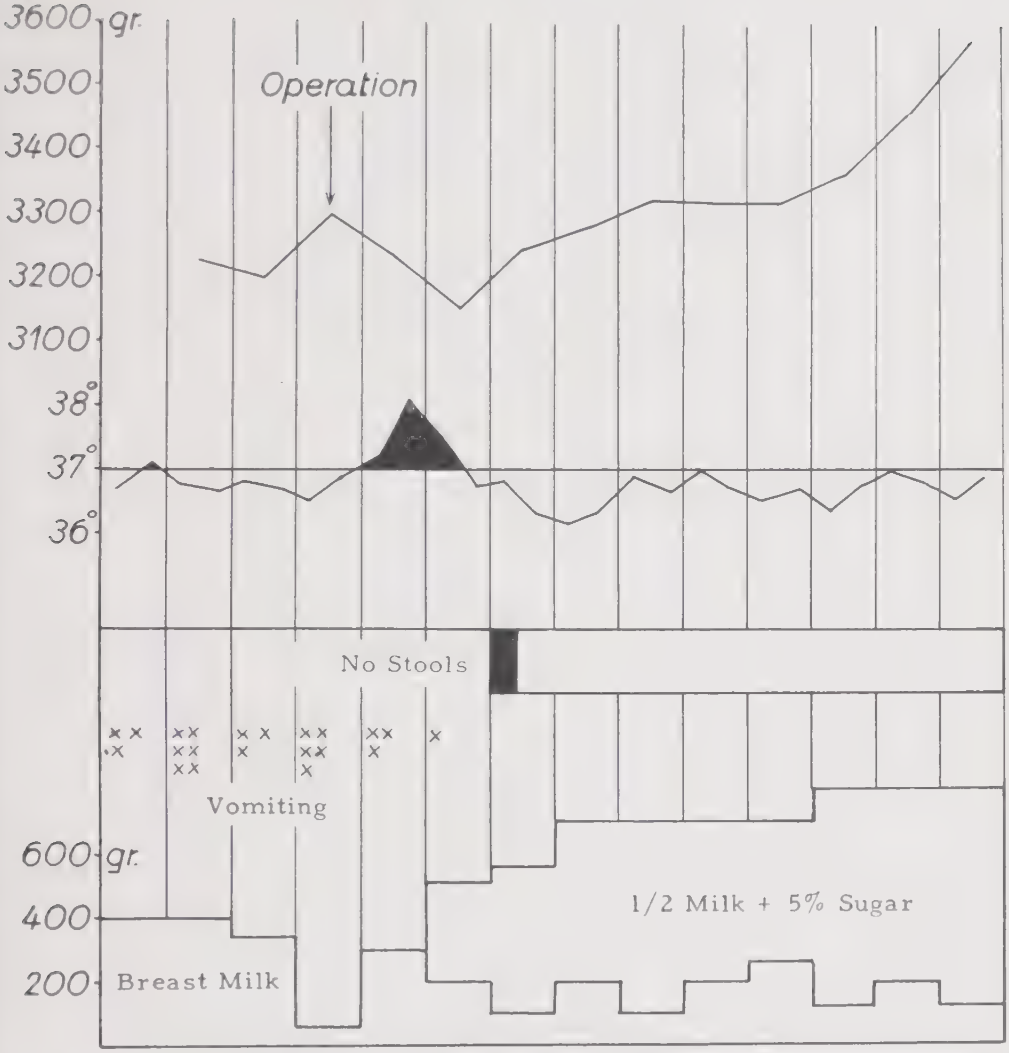

Fig. 28. Infant H.D., six weeks old. Birth weight 2800 g. Pyloric stenosis. Vomiting for three weeks. Constipation. Dystrophy. Surgery. Postoperative course: fever for two days, cessation of vomiting, good development.

operation. It is up to the surgeon to decide whether the operation should be performed under local anesthesia or general anesthesia (most surgeons recommend open drop method of ether). The child must be kept warm during the operation. The operation itself is not dangerous, losses of blood are small. It consists of a lengthwise splitting of the musculature of the hypertrophied pylorus. After cutting the peritoneum the muscles are severed with a dull instrument down to the muscularis mucosae. Particular care must be taken that the muscle is split completely. If even a few muscle fibres remain the success of the operation is disturbed. On the other hand the lumen of the stomach or the duodenum must not be opened. This mishap occurs particularly easily on the duodenal end of the hypertrophic pylorus which here is usually less sharply differentiated from the normal intestinal wall. Careful closing of an unintentional incision into the intestinal lumen is necessary. Any modification of the procedure increases the danger of the operation. This applies to wedge shaped excisions of the pylorus and attempts to cover the wound with the omentum. After the introduction of the *Weber-Ramstedt* operation the previously applied gastro-enterostomy has been abandoned completely.

As a result of the operation and anesthesia young infants frequently develop convulsions. Therefore every child receives upon termination of the operation immediately 0.05 to 0.1 Sevenal (Pheno-barbiturate).

For post-operative treatment most surgeons hand the child back to the care of the pediatrician. Care and nutrition of the child requires pediatric experience. Temperatures up to 39° occur regularly within the first 24 hours after operation. The origin of this fever is unknown. It has no connection with any changes in the operated area, peritoneal irritation, septic processes, etc. The fever disappears after 24 to 48 hours. After the operation the child receives no food for about four to six hours, not even liquids per os. If the child is severely ill and dehydrated, infusion should be repeated after the operation and possibly continued slowly for a number of hours. At this time, too, saline and glucose solutions or plasma are given. About four to six hours after the operation it is permissable to give the child, if it is awake, water sweetened with saccharine by teaspoon. Feeding can be resumed about 12 to 24 hours after the operation. The removal of the obstacles on the pylorus offer the temptation to give the emaciated infant immediately larger quantities of food. Feeding should, however, be started with small quantities, even if breast milk is available. It is a mistake to give children larger quantities of any type of food immediately following the operation or to put them on the breast of the mother. Toxic conditions can develop if children receive larger quantities of food immediately after the operation. Feeding is resumed after 12 to 24 hours at first with small quantities, 10 times 10g breast milk or sweetened buttermilk or half milk with 5% sugar. On the second day the food quantity is increased to 20 to 30g per meal, and then rapidly raised from day to day until the required quantity and composition of food is reached. If the post-operative course is undisturbed the child can be placed on the breast about three to four days after the operation. One must always remember, however, to meet the fluid requirements of the child, either by adequate water intake per os or by infusion as long as the natural or artificial food intake does not cover these requirements. Children whose nutritional state was already considerably damaged frequently recuperate with surprising rapidity after a successful operation. Vomiting ceases, although the tendency persists for a while to react to minor stimulations with renewed slight attacks of vomiting.

The dangers of operation lie mainly in faulty post-operative care. The normal rapid convalescing process is not infrequently interrupted by disturbances which must be recognized and treated in time. Atony of the stomach and accumulation of bilious bloody secretion in the stomach occur relatively rarely. The short duration of the operation, the absence of manipulations on the mesenterium explain the rare occurrence of otherwise frequent complications after abdominal operations during infancy. Atony of the stomach manifests itself through dilatation of the upper abdomen, vomiting or the flow of greenish-brownish substances from the mouth. Introduction of a stomach tube and rinsing of the stomach through the tube with small amounts of liquid usually removes the disturbance within a few hours. No feeding should be undertaken as long as the rinsing liquid does not flow back clear. The resumption of feeding in these patients should be handled with particular caution. In other patients vomiting continues even after the operation although the operation was technically faultless. By placing the child in a position whereby the head and shoulders are raised, and giving atropine or luminal, the remaining tendency to vomit can be removed.

Several days will pass until the food intake will be large enough to bring about a weight gain. Once this stage is reached the further development of the child is undisturbed. In exceptional cases (if the operation was not properly performed) vomiting continues. It might then be necessary to repeat the incision of the pylorus. Not rarely do we observe peritoneal adhesions and bands following the operation, which in one of the children under our observation resulted in the development of a secondary ileus.

If a child suffering from pyloric stenosis is cured through medical or surgical treatment, the prognosis for the further development of the child is good. Even in infants treated successfully by drug therapy the continued existence of a hypertrophy of the pylorus no longer impedes the gastric functions. After the third month of life the disturbing spasm in the hypertrophic muscle almost always disappears. After that time the development of a pylorospastic child does not differ from that of a healthy child. Even in later years and beyond childhood there are no differences between these patients and other children. Most important, diseases or disturbances on the part of the stomach are not more frequent in these patients in later years than in the average population.

CHAPTER VI

Nutritional Disturbances Due to Disturbances of Glandular Secretions

CONGENITAL HYPOTHYROIDISM

If the thyroid gland is absent or incompletely developed (hypothyroidism, athyroidism, myxedema) difficulties in food intake are frequently an early, at times even the first sign of disease. A more or less marked lack of interest in drinking or displeasure with eating which appears already in the first weeks of life must be all the more noticeable since difficulties of this type are rare at this early age. Feeding difficulties and refusal to eat occur much more frequently after the first half year of life. Any difficulties in food intake appearing at an early age must give rise to a search for symptoms of hypothyroidism. The pathological symptoms resulting from a lack of specific secretion of the thyroid gland are usually not present at birth. They begin to appear in the course of the first weeks of life. The skin becomes dry, the hair dry and sparse, the tongue is gradually enlarged until it cannot find room enough in the mouth. The typical dough-like swelling of the skin in myxedema appears relatively late, at first on the back of hands and feet, on the back, the forehead and in the supra-clavicular fossae. The skin is cool, livid or pale. The development of bone centers is retarded. Increased values for cholesterol and carotin are found in the blood, although not in every case. In addition, there is an obstinate constipation which cannot be cured even with cathartics and changes in the diet. All pathological symptoms, including constipation and anorexia, disappear within a few days after administration of thyroidin.

The earlier specific therapy is instituted the better is the prognosis for the later development of the patient. Therapy should be continued without interruption until an improvement in all symptoms can be seen, including the return to normal of the cholesterol content in the serum. Later on pauses of longer or shorter duration can be made between thyroidin medication. As initial dosage infants receive 0.05g of an effective thyroid preparation. This quantity can be increased to twice 0.05g. After the preparation has shown results it can be given during periods of eight to 14 days, with pauses of eight days between administrations.

ADRENO-CORTICAL INSUFFICIENCY

Already during infancy adreno-cortical insufficiency can result in feeding problems. Children with insufficient secretion of the adrenal cortex suffer from vomiting which resembles that observed in pylorospastic children. Stools are infrequent. The children develop poorly; they are dystrophic. The skin assumes a golden-brown, later brownish and even darker coloration. Fainting spells and convulsions occur. The diagnosis of insufficiency of the adrenal cortex is proven by examination of the blood serum. The sodium content of the serum is low, potassium content elevated, and the blood sugar curve is flat after sugar intake. The cause of the disease is a disturbance in the functions of the adrenal cortex, among which are the regulation of the sodium chloride fixation and the potassium elimination through the urine. The functions are regulated by the 11-desoxycorticosteroids of the adrenal cortex. The adrenal cortex plays an important role in sugar metabolism which is regulated by 11-desoxy-17-hydroxycorticosteron (S = sugar) hormone). In addition other corticosterons (so-called N-hormones) exert an influence on the nitrogen metabolism.

For the treatment of the disturbance adrenal-cortical extract (DOKA + vitamin C, cortisone) can be tried. In addition, however, sodium chloride and, in order to prevent hypoglycemia, glucose must be given. The improvements which can be obtained in this manner are, however, only temporary. The prognosis of congenital or early appearing adreno-cortical insufficiency is poor.

FIBROCYSTIC DEGENERATION OF THE PANCREAS

(Congenital pancreatic fibrosis) was formerly not differentiated from the celiac syndrome because of certain external similarities. Pancreatic fibrosis is an anatomical pathological entity, frequently familial, recessive, which causes chronically retarded development and digestive disturbances. Larger or smaller parts of the excretory section of the pancreas show cystic degeneration which is in most cases congenital. Insulin production is not disturbed. The disease is frequently accompanied by disturbances in the bronchial tree. The anatomical picture of congenital pancreatic fibrosis is a cystic enlargement of the excretory ducts and glandular parts. The epithelium of the gland degenerates and its place is taken by increased connective tissues. Likewise we find in the bronchial tree bronchiectases, purulent bronchitis, thickening of the bronchial wall, metaplasia of the epithelium and an enlargement of the mucous glands which, similar to the excretory ducts of the pancreas, are filled with viscid secretion. The combination of the two symptoms: cystic degeneration of the pancreas and metaplasia of the epithelium in the bronchial tree can be explained by the developmental history of both organs from the same germinal layer and the same "Anlage." The particular viscidness of the secretions of both organs causes an obstruction of the excretory ducts. The accumulation of the secretion leads to cystic degeneration of the pancreas or the dilatation of the lowest sections of the bronchial tree, respectively.

The earliest manifestation of the disease is *meconium ileus.* The newborns fail to eliminate meconium. A sausage-shaped, large, soft tumor can be palpated in the distended upper abdomen. The child vomits meconium and if not treated, dies after six to eight days of hunger, with the symptoms of ileus or a rupture of a section of the intestines, filled to the utmost. The normal alterations of the intestinal content do not take place due to the absence of pancreatic secretion. Trypsin, which liquefies the protein content of the meconium, is lacking. The meconium is of a different colloidal composition than the normal intestinal content of the newborn. An unusual mixture of protein, fat and water produces a sticky, viscid mass which sticks to the intestinal wall and cannot be moved further (Dysporie *Glanzmann*). Attempts to liquefy these masses of meconium by administering pancreatic enzymes per os, to make them softer so that they can be transported, are successful in only a few cases. The administration of prostigmin, for the purpose of removing the ileus, is dangerous as it accelerates the rupture of the intestine. In a few patients it has been possible to empty the small intestines surgically through irrigations. The sections of the intestinal tract which lie beyond the tough masses of meconium frequently are so narrow that normal peristalsis cannot take place even after emptying of the intestines and medication of pancreatic enzymes.

After the newborn period the disease manifests itself in form of a chronic digestive disturbance with bulky, fatty, foul smelling, sticky stools. The abdomen of the child becomes distended. Development of the children ceases. Even with a diet which is poor in fats the fat content of the stools is four times higher than normal. About half of the ingested protein and 15% of the carbohydrates are lost in the stools. The children assume a greyish appearance. Hypochromic anemia develops as well as hypoproteinemia and hypoglobulinemia. Thus the basis for the development of hydrops is given. The pulmonary symptoms vary in their intensity. While many children suffer simultaneously with the development of the digestive disturbances from constant whooping-cough like cough or recurrent bronchitis and pneumonia, other patients show only minor pulmonary symptoms so that the condition resembles even more that of celiac disease. The diagnosis of cystic degeneration of the pancreas is confirmed by proving the absence of external secretion of the pancreas (trypsin, lipase). This is determined by examination of the duodenal secretion, obtained through insertion of a duodenal tube for trypsin and lipase, respectively. This method produces the clearest results. Insertion of a duodenal tube in the infant is a difficult procedure which requires much patience. It is simpler to make a probable diagnosis by microscopic examination of the stools after intake of whole milk (without simultaneously giving pancreatic enzymes). If fat droplets are seen in the stool the fat digestion is inadequate (lack of lipase). The absence of trypsin can be proven by diluting small quantities of stool with distilled water in a relationship of 1:5 or 1:10 respectively and placing it on the gelatine side of an undeveloped small piece of roentgen film (after removing the gelatine from the other side of the film) for one hour in an incubator at a temperature of 37°. The film is then rinsed with cold water. If trypsin is contained in the stool the film is clear and transparent in both places (1:5 and 1:10

dilution). In the case of cystic degeneration of the pancreas the digestion of the gelatine is incomplete or lacking altogether. The test must, however, be repeated two to three times. Since enzymes produced from certain intestinal bacteria, proteus, pyocyaneus, have trypsin-like actions, a stronger dilution of the stool (1:100) should always be used in order to avoid such errors. With these strong dilutions the trypsin effect of bacteria is eliminated.

The prognosis of the disease is always doubtful. A cure is not possible and the danger of pulmonary diseases is great. The disease requires constant treatment. Therapy consists of 1) continuous medication of pancreatic enzyme preparations (Pancreatin etc.) in large quantities (0.1–0.2g) with every meal, and 2) a diet containing no fat but large quantities of protein (6 to 8g per kilogram bodyweight). It is advisable to give part of the protein in form of hydrolyzed casein (1 to 1.5g per kilogram bodyweight), although a number of vital amino-acids are lacking in hydrolyzed protein preparations. To meet the requirements of fat soluble vitamins older patients receive daily an egg yolk. It is important to cover the vitamin A requirements since the lack of this vitamin might perhaps play a part in the development of pulmonary changes. The pulmonary changes and the accompanying secondary infections are treated with penicillin.

True celiac disease can be differentiated from cystic degeneration of the pancreas by the absence of pulmonary changes, the presence of pancreatic enzymes in the duodenal juice and the proof of trypsin in the stool.

CHRONIC AMINO-ACIDURIA

(Renal rickets with amino-aciduria, glycemic dwarfism with amino-aciduria.) Chronic amino-aciduria is a rare, recessive hereditary disturbance of the amino-acid metabolism, whereby the re-absorption of amino-acids from the proximal part of the tubuli is deficient due to a lack of phosphatase. A special type of amino-aciduria is the cystine disease, the cystinosis of infants, whereby cystine is deposited in the cells of the reticulo-endothelial system since the ability to eliminate cystine through the kidneys is reduced. The disease develops in the second half of the first year of life. The symptoms are: lack of development, dwarfism, changes in the bones resembling rickets (so-called renal rickets), polyuria, isostenuria and in most cases glycosuria, albuminuria and phosphaturia. The urine reacts neutral or more frequently alkaline, probably as a result of the strongly increased ammonium elimination and the elimination of not re-absorbed alkaline amines. Blood findings include hypophosphatemia with normal calcium values and a tendency to hypocholesterinemia, a low alkali reserve, variable values of the blood sugar with a tendency to hypoglycemia. Children develop thermolability (fever with thirst) polydipsia and a tendency to conditions resembling intoxication, particularly after large and rapid intake of dextrose (sugar shock). Occasionally cystine crystals can be found in the urine or the bone marrow.

The prognosis of the disease is serious in characteristic cases. No effective therapy is known. A diet rich in alkalis as well as medication of calcium and phosphates has been recommended.

CHAPTER VII

Qualitative Nutritional Disturbances

Any lack of one or more vital substances sooner or later shows effects upon the development and functions of the body of the infant. Similar disturbances develop if an important nutritional substance is not contained in the food in adequate quantities. It is one of the peculiarities of the rapidly growing infant that the organism reacts much sooner and much stronger to any qualitative deficiency in the food than in the slowly growing older child or in the adult. Qualitative nutritional disturbances are therefore a very frequent occurrence during infancy.

Frequently the lack or insufficient supply of one substance is accompanied by a deficiency of a second or even a number of other food components. This causes "nutritional disturbances" which present a great variety of symptoms. The nutritional disturbances will be discussed under the name of the essential substance which is deficient.

NUTRITIONAL DISTURBANCES DUE TO LACK OF PROTEIN

A protein deficiency hardly ever occurs if an infant is fed with the customary milk mixtures since even the strongest dilutions of cow's milk which are occasionally still recommended, with two parts water and one part milk, still fully cover the protein requirements of the young child. An undersupply of protein is more likely to occur in breast fed infants (see p. 102) who occasionally might be dependent on an increased protein intake either due to unusually rapid growth or because they might be unable to replace protein losses, resulting for instance from prolonged infections, due to the low protein content of breast milk. This includes rapidly growing prematures and infants convalescing from severe nutritional disturbances. By the addition of protein preparations (casein-calcium, amino-acid preparations) the dystrophic conditions of these patients can be cured.

In recent years cases have been reported in which a hypoproteinemia, resulting from extreme protein deficiency in the food, led to severe symptoms characterized by complete loss of fat, edema and changes in the skin. In these children the hair becomes thinned, depigmented, reddish. In the skin pigment anomalies (depigmentation) or increased pigmentation develop which might perhaps be the result of a simultaneously occurring vitamin C deficiency. Other symptoms of avitaminosis (xerophthalmia, pellagra-like skin changes) are not infrequent. Malnutrition leads to ulcerations of the skin which resist treatment. The liver of these children shows fatty degeneration and infiltration. The adrenal cortex is atrophic. These conditions, whereby a protein deficiency in the food is accompanied by multiple hypo- or avitaminosis, have been referred to as Kwashiorkor, malignant malnutrition, fat-liver disease etc. The disease can be cured by adequate intake of animal protein or egg yolk. Egg white does not improve the disease.

Furthermore, conditions which are referred to as "protein deficiency diseases" (*Fanconi-Prader*) have been described which can be classified about half way between the serious conditions connected with hypoproteinemia and the damages due to excessive flour intake (Mehlnährschaden) which will be discussed below. A protein deficient diet leads to dystrophy, a lowering of the protein values in the serum and to anemia. In children all symptoms accompanying a severe dystrophy (hydrolability, tendency to develop severe gastro-enteritis, etc.) are present. Infusions of protein-rich solutions (plasma, amigen) and a protein and vitamin rich diet which is also given in case of diarrhea, cure the disease.

All these syndromes (Kwashiorkor, malignant malnutrition, protein deficiency dystrophy) are closely related to the symptoms caused by excessive flour intake (*Czerny*) which have been known for a long time. Kwashiorkor differs from damage due to excessive flour intake through anomalies in pigmentation and changes in the skin ("crazy pavement"). These and other secondary symptoms develop if there is a simultaneous deficiency or lack of one or the other vitamin of the B complex or of vitamin C, which produce additional symptoms to those of the protein deficiency diseases.

Protein deficiency, together with a lack of other important substances, develops in those infants who are for a certain period of time fed exclusively with cereals (flour soups, rice water). The resulting symptoms have been referred to as "damages due to excessive flour intake" (Mehlnährschaden).

Today damage caused by excessive flour intake is a rare occurrence, of only historical interest, thanks to the progress made in child care and to the education of mothers and nurses. A short description of these damages is, however, justified since it can serve as an example for all nutritional disturbances. Damages caused by excessive flour intake demonstrate most clearly the serious consequences of a qualitatively inadequate nutrition. There are not only disturbances in growth but the entire functions and development of the youthful organism are severely affected over a long period of time.

Damages due to excessive flour intake develop when infants are fed over a longer period of time on a diet consisting essentially of boiled flour or flake preparations which might also be sweetened with sugar and which, in accordance with the customary recipes, also contain salt in relatively large quantities. All boiled flour mixtures are rich in carbohydrates and water absorbing cooking salt. They lack protein, fat, vitamins and calcium.

This type of incomplete diet is occasionally still recommended today, for instance in form of rice water, as diet in the treatment of diarrhea, particularly dysentery. After the diarrhea improves, the mother, fearing a recurrence, is tempted to continue the one-sided cereal diet for weeks, even months. In a few countries there still exists the custom that when the flow of breast milk ceases young children are fed with flour, bread and similar foodstuffs over longer periods of time. The speed with which nutritional disturbances develop on such a diet depends on the age of the child. The rapidly growing cells of the youngest infants with their great swelling ability suffer much more under a faulty diet than the cells of the older infant whose growth impulse has already diminished.

The symptoms of damages developed due to excessive flour intake are those of dystrophy or atrophy, except that there is an initial spurt in weight gain in the first stages of the disease due to the effects of the excessive carbohydrate intake and the above normal swelling of the tissues (pseudo-edema). However, the physiological firmness in the formation of the tissues is lacking. The body resembles a sponge which absorbs large quantities of water. Similar to the sponge, where little pressure is sufficient to remove the loosely held water, the inordinate weight gains in the flour-fed infants disappear from any minor cause. The "apparent growth" is invariably followed by a reversion.

While the waterlogged condition persists the appearance of the children is not bad. However, the pink color soon fades and the children become pale. The face appears swollen. Edema develops on eyelids, the lower legs and on the forehead. Inflammatory kidney disease is suspected but the urine fails to reveal protein, cylinders etc. The turgor of the tissues diminishes. The resistance of all tissues is reduced. Stubborn furunculosis appears on the waterlogged skin. Grippal infections occur more frequently than in the healthy child.

The presence of diarrhea completes the symptoms caused by excessive flour intake. The lack of vitamin C causes scurvy-like symptoms. Vitamin A deficiency results in keratomalacia.

> In the various organs of children who had died after prolonged flour diet a water accumulation in skin and musculature was found as well as an increase of chlorine and sodium at the expense of potassium and phosphorus. The water content of the blood is elevated up to 85 and 86%, as compared to the normal percentage of 80. The loss of mineral substances can, after prolonged flour diet, reach a stage where the elimination of hydrochloric acid in the stomach ceases and no chlorine is eliminated in the urine. These findings permit no conclusions regarding the colloidal changes in the tissues.

The diet chosen in the treatment of damages caused by excessive flour intake must avoid the development of diarrhea, must contain all substances necessary for rebuilding the tissues and must also re-establish the lost immunity. In the severest forms of the disease and in young infants during the first months of life these requirements are best met by feeding the patient with breast milk or one of the special infant milk preparations (protein milk).

The quantity of breast milk, with the addition of protein preparations, depends on the condition of the intestinal tract. If there is no diarrhea larger quantities of breast milk covering the requirements of the infant can be given from the beginning. Therapy is initiated by giving 300 to 400g breast milk per day, divided into five to six meals. If no breast milk is available or if the child suffers from diarrhea treatment should proceed along the lines prescribed in cases of acute diarrhea (see p. 353). The first reaction to the change in diet in children suffering from damages due to excessive flour intake is a rather considerable weight loss caused by elimination of excess water which was stored, but only loosely absorbed, in the body.

By feeding the child exclusively with breast milk the period of recovery is long, even if the caloric requirements of the child are amply met, since there is a relative deficiency in body building nutritional substances. The child will show a gain in weight only after other foodstuffs (proteins, vitamins) are added to the diet. Breast milk or a special infant food (buttermilk with fat added, protein milk) should be continued over a longer period of time if the child was very young or if the damages caused by the excessive flour intake were extensive and severe. Dystrophic children will require about one or two months for recovery, while atrophic infants might require two to three months and more.

In children beyond three months of age treatment of severe damages is effected with artificial diets. Artificial feeding offers the opportunity to choose an infant food which prevents the development of diarrhea and which removes the protein deficiency by an ample protein content. Food mixtures suitable for the treatment of these children must contain protein, minerals and vitamins in adequate quantities. These requirements are best fulfilled with buttermilk, protein milk or, in patients with little disturbances of the intestinal functions, with sweetened whole milk.

As soon as the condition of the intestinal tract permits fat must be added to the diet. The diet must further on be complemented as early as possible by the addition of fresh fruit juices in quantities of 50 to 80g daily, even when the stools are still frequent and soft. Bananas and egg yolk are important since they contain vitamins B and A. Even with adequate nutrition it takes a considerable length of time before the lost immunity is regained.

In infants beyond the ninth month of life, who have been damaged by excessive flour intake, the diet should be supplemented in many directions. The particular likes and wishes of the child can be considered to a large extent in choosing the diet. puréed meat, fish, egg, bread and many other types of food can be given in the dietetic treatment. The fat deficiency of such a diet can be complemented by the addition of butter or margarine, possibly in form of roasted flour.

NUTRITIONAL DISTURBANCES DUE TO LACK OF CARBOHYDRATES

(Damages Caused by Excessive Milk Intake (Milchnährschaden))

Children who are fed over a long period of time exclusively with large quantities of milk with no, or only little sugar added, fail to show a weight gain even though their food intake is calorically adequate, frequently even above the required amount. A number of characteristic general symptoms appear in these children who lack in development: pallor of the skin, loss of tonus, decrease in turgor, reduction in immunity, signs of general discomfort and irritability and as outstanding symptom, the elimination of fatty soapy stools, that is, stools of a whitish or greyish color, crumbly and with a low water content, which do not adhere to the diaper and which, because of their light color, apparently contain no bile pigment.

Dystrophic conditions develop without, however, showing the severe changes in the tissues which are characteristic for damages caused by excessive flour intake. Damages caused by an oversupply of milk never lead to atrophy. The milk, with no or only little sugar added, supplies the child with all necessary food components. Therefore, there is only a relative deficiency of sugar. The quantities of sugar contained in whole milk suffice for a number of children, so that some infants show a satisfactory development despite exclusive milk feeding: Only in certain children will this type of nutrition produce pathological symptoms.

Metabolic tests revealed that the protein metabolism remains undisturbed and that the utilization of fat is not reduced at all or reduced to a minor degree only. However, there are considerable changes in the composition of the fecal fat: the unsoluble calcium and magnesium soaps are increased to almost three times the normal amount. The changes are the result of an inadequate relation between fat and carbohydrate. In whole milk and in unsweetened milk dilutions the correlation of fat to carbohydrate is 1:1. The infant suffers from a relative lack of carbohydrates since for the majority of infants the relation of fat to carbohydrate must be 1:2. In this form of nutritional damage the cause for the disturbance in development lies in the incorrect correlation of fat to sugar, the antagonists in the metabolic processes, and not in the milk fat itself.

Carbohydrate deficiency manifests itself in two directions. The carbohydrate requirements of the growing cells are not met and abnormal chemical processes develop in the gastro-intestinal tract. The small quantities of carbohydrate contained in whole milk are rapidly absorbed from the small intestines and already at the beginning of the large intestines the chyme is practically free of carbohydrates. The stimulus on the peristalsis, which originates from the fatty acids formed from carbohydrates, is lacking. Peristalsis is slowed down so that the food remains longer in the intestines and as a result the water in the food remnants is completely absorbed. The stools which are eliminated contain only very little water, they react alkaline and are hard and usually malodorous and are referred to as fat-soap-stools. The light color of these stools is also a result of reductive processes of the biliary pigments due to the slow passage of the food remnants through the large intestines; these processes might be facilitated by the alkaline reaction and possibly by special types of bacteria.

Symptoms of dystrophy resembling those caused by excessive milk intake can be observed also in children beyond the sixth month of life; they develop in cases where exclusive milk feeding is continued beyond the physiological period. This form of dystrophy, with symptoms of damages caused by an oversupply of milk, occurs not infrequently in prematures and in weak infants where as a result of a delay in physical development timely addition of other foodstuffs is unjustifiably feared and therefore omitted or refused by the children. In these patients we find not only a relative carbohydrate deficiency but also a lack of nutritional substances which are contained in the mixed diet which an older child requires. Some of the vitamins are lacking. In these cases it is not only the carbohydrate deficiency which causes the symptoms occurring with excessive milk intake, but also the absence of other foods.

In younger infants with light, uncomplicated symptoms, it is sufficient to add sugar or to increase the sugar in the food to 6, 8 or up to 10% of the food quantity, in order to eliminate the disturbances in growth and in the stools. In children with less damage or with constipation the sweetened milk mixture can immediately be given in a quantity which corresponds to age and weight of the child.

In children with more severe damages who already show a tendency to diarrhea, part of the sugar is replaced by flour or, in children during the first months of life, by cooked cereal water. Caution is indicated particularly in those children who had previously already suffered from diarrhea. For such cases buttermilk with 5% sugar and 1 to 2% flour is recommended. These mixtures provide the amounts of carbohydrate necessary for treatment. If there is no diarrhea quantities corresponding to the age and weight of the child can be given from the beginning. If diarrhea had occurred shortly before or if the stools are still liquid, the sweetened food should be limited, after a short interruption during which only tea is given, to about 300g per day which can, however, be increased rapidly from day to day until it covers the requirements of the child.

Most effective carbohydrate intake, with simultaneous decrease of milk fat and milk protein, is available in form of *Keller's* Malt Soup which formerly was widely used, particularly in infants beyond the third month of life. Malt soup, however, is incomplete in its composition as an infant food and in its correlations deviates considerably from the norm. It should, therefore, be replaced soon by a food which fulfills all the requirements of the growing organism. *Keller's* Malt Soup has today disappeared almost entirely from the diet of infants.

Even in prematures and in children whose development is delayed, unjustified fear of the development of pathological intestinal symptoms should never result in the omission of necessary additional foods (fruit and vegetables).

The prognosis of damages caused by excessive milk intake is good, although intercurrent infections threaten the children because of their lowered immunity. Decrease in immunity particularly affects the skin. Furuncles and abscesses are not infrequent. The return to an eutrophic condition can be accomplished in a relatively short time. Damages caused by an oversupply of milk are among those diseases which can always be avoided by adhering to the simple rules of artificial nutrition.

WATER DEFICIENCY OF THE DIET

While the nutritional disturbances which have so far been discussed develop slowly and only gradually affect the development of the child, disturbances resulting from water deficiency in the diet appear only rarely in this slowly progressing form. In such cases we usually observe acute symptoms which take a violent course.

Water deficiency can develop in the following ways:

1) Through insufficient water intake on a diet consisting of the customary feeding mixtures.
2) In diets consisting of concentrated feeding mixtures, particularly rich in protein.
3) Through water losses through regurgitation and vomiting.
4) Through water losses on hot days.
5) Through increased demands on the water metabolism in infectious fevers.

Lack of water is a deficiency disease which can be cured by supplying water, the food component which hitherto had been given in insufficient quantities.

The danger of water deficiency is particularly great in the first half year of life since the water requirement of the infant are unusually high, amounting to an average of about 150g per kilogram of body weight per day. This does not mean, however, that the average requirements which are obtained by most breast fed infants, are necessary in every case for the development of the child. Water requirements vary considerably in different children (80 to 200g per kilogram of body weight per day) depending on constitutional characteristics of the child. Many individuals can therefore get along on less than 150g per kilogram of body weight per day. Development with relatively small quantities of water might perhaps point to a better general constitution with regard to formation and solidity of tissues.

The high taxation of the water metabolism during infancy explains the fact that these disturbances occur much more frequently in the earliest period of life than in the older child or the adult. Because of the very large body surface in relation to weight, because of the active metabolic processes, and because of a diet which is relatively high in proteins, relatively much larger quantities of water are required for metabolic processes and for elimination than in the adult. The kidneys, which are rather large in relation to the body weight (about 0.75% of the body weight in the infant and 0.5% in the adult) are a morphological expression for this activity. The edema formation which is much stronger in the child as compared to the adult and which can develop from a number of minor causes, and the tendency to rapid large water losses and to conditions of dehydration which in later years appears only during most serious diseases, demonstrate the peculiarities of the water metabolism in the infant. The extracellular fluid which essentially provides for the water metabolism, amounts in an infant of three to four kg to about 700cc. The fluid intake amounts to about 700 to 800g, the output in urine, stool and insensible perspiration to also about 700 to 800g, that is, an infant metabolizes about 100% of his rapidly disposable extracellular fluid in the course of a day. A similar calculation for the adult shows a metabolism of only 15 to 20% of his extracellular fluid.

There is, however, a lower limit for all infants below which the water intake must not drop if pathological symptoms are to be avoided. The minimum water supply amounts to about 75g per kilogram of body weight per day. With the customary milk mixtures no water deficiency will develop so long as adequate amounts are offered and taken by the infant. The possibility exists only in the first days after diarrheal diseases when a special diet is given. The water requirements must then be covered by supplying it in some other form (per os, parenterally).

With absolute or relative water deficiency in the diet the infant beyond the newborn period develops acute or subacute pathological symptoms which appear within a few days, even within hours. Signs of water deficiency are: hyperthermia, weight losses and a number of signs of disturbances in the general condition which develop more or less suddenly depending on the degree of damage.

Water deficiency develops in young infants during the first week of life when the milk secretion of the mother is inadequate or ceases altogether and the child is not supplied with the necessary amounts of liquid in form of tea or water sweetened with saccharine or sugar. The children become restless, the skin dries out, the mucosa of the lips becomes red and cracked, breathing becomes more rapid. Body temperature rises to 39° and higher.

Somewhat older children who fail to receive an adequate amount of fluids at first appear pale and grey, with rings around the eyes, the lips are bright red, cracked and dry. The mucosa of the mouth shows similar changes. The fontanel is sunken, the skin withered, dull and dried up. The children are restless and prolonged crying increases the water losses through lungs and skin. Occasionally a pre-edema conceals the large water losses. Despite this fluid which is still retained in the wrong place the water losses which have already occurred in the tissues must not be overlooked. These conditions are observed particularly in newborns in whom the tendency to develop edema is especially strong.

Similar symptoms of dehydration can be observed on the inner organs. The elimination of protein, leukocytes and cylinders in the urine of thirsting children is a direct result of the dehydration of the urinary passages (e.g., pyuria of dehydration in newborns and infants with severe pylorospasm). This probably includes also the so-called physiological albuminuria of newborns in the first days of life. If generalized convulsions and somnolence occur the condition becomes alarming.

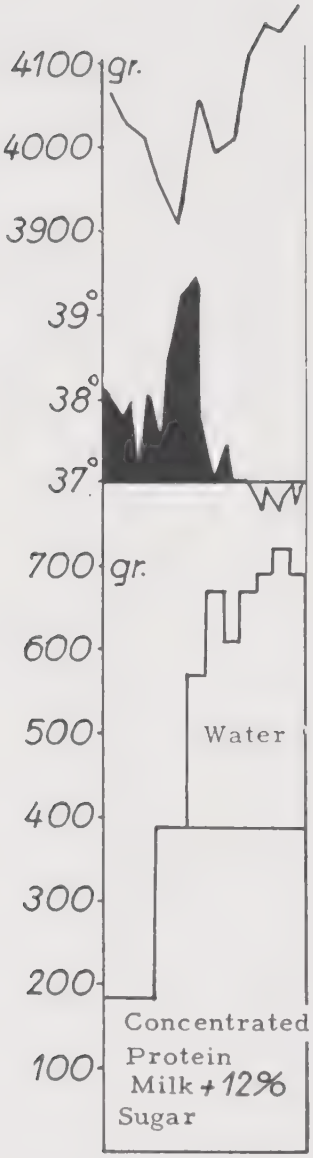

Fig. 29. Infant S.R., two and one-half months old. Fever due to thirst. High fever caused by feeding concentrated, protein-rich foods. Return to normal temperature effected by addition of water to diet.

Similar serious conditions develop in the infant only in the course of infections which are accompanied by violent vomiting or when the child refuses to take food. Dehydration develops which is observed in cases of severe acute nutritional disturbances (toxic gastro-enteritis, see p. 342).

The subacute or chronic form of water deficiency is frequently not recognized. The alarming symptoms of the damages caused by thirst are absent. A subacute state of water depletion develops particularly in premature children and young infants during the first weeks and months of life. The essential sign of water deficiency is the absence of weight gain despite calorically adequate, even ample nutrition. By addition of protein in some form the demands on the water metabolism increase. Water requirements are greater in artificially fed infants than in children raised on breast milk which has a relatively low protein content. After a while dystrophy develops. Occasional temperature elevations, possibly a slight pallor and dryness of the mucosa, the appearance of protein and leukocytes in the urine, hardly suffice to draw special attention to the absence in weight gain. The addition of water reveals the nature of the disturbance. Weight gain ensues without any other changes in the diet.

Danger of water deficiency exists always in diets consisting of feeding mixtures which contain double or triple the amount of calories of the customary milk mixtures (concentrated food). While the nutritional requirements of the child are covered the food quantities contain only one half or one third of the water quantities required. If a child is fed exclusively with concentrated feeding mixtures or cereals—as recommended for instance for vomiting children—the fluid requirements of the child are not covered unless fluid is given after or between meals in form of weak tea or boiled water.

Infants who vomit frequently lose not only nutritional substances but also considerable quantities of water, particularly in the state of nausea. Damages due to thirst occur therefore not infrequently in children who regurgitate or in children with pylorospasm, particularly since concentrated diets are ordered at times for therapeutic reasons.

> The symptoms developing with a diet rich in proteins and salts resemble those which we observe in thirsting children. Two theories have been advanced to explain the high water requirements on a diet abundant in protein. One theory traces this occurrence back to the increased digestive activity in the breakdown of protein in the body (specific dynamic action of protein), which results in an increased heat production in the body. By an augmentation in insensible perspiration the body is able to remove the excess of heat provided sufficient quantities of fluid are at its disposal. If the necessary amount of water is not present, a state of thirst develops.
>
> The other theory assumes that water is necessary for the absorption of protein and for the removal of metabolic waste products from the tissues. For the elimination of the waste products of protein relatively large quantities of water are needed. If in a high protein diet the quantity of protein waste products increases, part of the waste products cannot be eliminated through the urine unless the water intake is increased accordingly. The accumulation of such protein waste products, which remain in the tissues, damages the cells and causes secondarily symptoms of a state of thirst.

The occurrence of the so-called transitional fever of the newborns which develops not infrequently in breast fed infants during the first days of life has been explained in a similar manner.

On hot summer days or in overheated rooms symptoms of water depletion can develop under inadequate care, if water is lost through strong perspiration, if the children lie in poorly ventilated, closed rooms. These water losses, resulting from increased insensible perspiration, have the same consequences for the entire water metabolism as insufficient water intake under normal nursing conditions. Children who are fed with concentrated feeding mixtures during the hot summer months are particularly endangered and therefore require constant and special care. In such instances it is of utmost importance that ample amounts of fluid are added to the diet.

Any temperature elevation increases the taxation of the water metabolism. The great thirst of feverish patients is the expression of the increased water requirements. These increased fluid requirements are not always covered, particularly in the infant whose state of thirst must be recognized in the beginning by not very characteristic clinical symptoms. It therefore happens not infrequently that an infant with temperatures which were primarily caused by an infection continues with a secondary fever which is no longer caused by the infection but which is a symptom of the water deficiency which had developed in the meantime.

Even in these cases the symptoms of water deficiency are not easily recognized. Only the success obtained with enteral or parenteral fluid supply shows the extent to which the water deficiency contributed to the high temperatures which were incorrectly ascribed to the infection.

Treatment of water deficiency depends on the degree of damage. In children who show only slight pathological symptoms and who are in a good nutritional state it is sufficient to give per os water sweetened with saccharine or sugar. Particularly in cases of fever due to infections it is advisable to combine the water intake with the addition of carbohydrates since protein catabolism can be decreased in this manner. At the same time those substances which like protein and salt place particular demands on the water metabolism should be limited temporarily. "Carbohydrates are the best guide for the water on its course through the circulation to the cell." The sweetened water is best given in from of a 5 to 10% solution, in amounts of 150g per kilogram of body weight but not exceeding 3/4 to 1 liter at the most per day. Subcutaneous, rectal, intravenous or intraosseal supply of fluid is indicated in cases of alarming water deficiency, of vomiting and in children refusing all nourishment.

The treatment shows immediate success in patients whose pathological symptoms were caused by water deficiency. The general condition improves within a few hours. The grey pallor disappears, the skin shows a better blood circulation and a better hydration, the restlessness ceases and is followed by a quiet sleep. The most impressive changes in relieving the water deficiency occur in the reactions of the body temperature. The temperature which previously had risen to 39° and more, drops abruptly. Only in cases where water deficiency was accompanied by an infection which was not yet cured the temperature will not drop to normal but will be reduced by one or two degrees and the infectious disease can then take its normal course without the additional damages caused by thirst. Frequently, however, a surprising temperature drop explains the situation. A temperature elevation which had hitherto been regarded as the consequence of a still present infection and for the existence of which some kind of secondary complication had been sought in vain suddenly disappears. The cause of the temperature was water deficiency while the infection had already been cured.

AVITAMINOSIS AND HYPERVITAMINOSIS

The complete, "classical" pathological symptoms of avitaminosis, caused by insufficient intake of one of the known vitamins, which can at any time be produced in the laboratory in animal experiments are seen very rarely by the physician, in times of great need or under particularly unhygienic conditions. Much more frequent are conditions which develop due to an inadequate vitamin intake, in form of insidious, abortive forms of the disease (hypovitaminosis). In addition there are diseases which are caused not by a lack but by the oversupply of certain vitamins and which are referred to as hypervitaminosis.

By offering an adequate diet, complete in every respect, it is possible to avoid the development of clear-cut, easily recognizable and definite stages of avitaminosis. A complete diet also prevents the development of dystrophic conditions of hypovitaminosis which precede the avitaminosis and which can be recognized by only a few and less characteristic symptoms.

The knowledge of the existence of these nutritional disturbances came through practical experience and animal experiments with beri-beri, scurvy, etc. Only the experiences in the laboratory uncovered the knowledge which makes it possible for the physician to recognize, treat and cure these nutritional disturbances.

The term "vitamins," according to *Friedrich v. Müller,* refers to "those substances which are essential to the body for maintain-

Food Substance	A *Antikerato-malacia*	B	C *Antiscurvy*	D *Antirickets*
Bread	—	+	—	—
Liver	+ + +	+ +	?	+
Kidneys	+ +	+ +	+	+
Brain	+	+ +	?	+
Fish	+	+	+	+
Fresh milk, raw	+ + +	+ +	+ + (variable)	
Powdered milk	+ + +	+ +	+ (variable)	
Skimmed milk	+	+	+ (variable)	
Buttermilk	+	+ +	+ (variable)	
Cream	+ + +	+ +		
Butter	+ + +	—	—	+
Cheese	+ +	+ +	—	
Egg yolk	+ + +	+	—	+
Tomatoes, raw	+ +	+ + +	+ + +	
Cabbage, raw	+ +	+ + +	+ + +	
Carrots, raw	+ + +	+	+	
Cauliflower	+	+ +	?	
Walnuts		+ +		
Head lettuce	+ +	+ +	+ + +	
Potatoes, boiled	+	+ +	+ + +	+
Spinach	+ + +	+ + +	+ + +	
Kohlrabi	—	+ +	+ + +	
Apples	+	+ +	+ +	
Bananas	?	+	+	
Grape juice	?	+	+	—
Lemon juice	—	+ +	+ + +	
Orange juice	+	+ +	+ + +	
Raspberries, raw			+ + +	
Peaches	+ +	+	+ +	

ing its organs and functions, for growth and for reconstruction, and which the organism itself cannot synthetize from other basic substances." The existence of such substance has been suspected already many decades ago by *Bunge* and *Forster* when they failed to keep test animals alive with synthetic feeding mixtures which contained all the food components which were then known. The full recognition of the significance of vitamins is based on the studies of the Dutch physician *Eykmann* on beri-beri. This knowledge has rapidly increased since that time and today we can distinguish between individual vitamins or groups of vitamins which are referred to as vitamins A, B, C, and D. In addition there are a number of other substances showing characteristics of vitamins such as vitamins K, E and P, which need only brief mentioning in the framework of a presentation of physiology and pathology of infant nutrition. Vitamins A and D (as well as vitamins E and K) are fat soluble. The vitamins of the B-complex and vitamin C are water soluble.

The preceding table presents a survey regarding the vitamin contents of some of the more important foods.

The chemical structure of the nature of most vitamins the existence of which could formerly be assumed only on the basis of their effects is today known and many of the vitamins are now synthetically produced.

VITAMIN A

A-Avitaminosis. Dystrophia alipogenetica. Keratomalacia

Vitamin A is formed in the liver from yellow-red substances of certain plants and fruits, the carotins, which are stored in the fats directly or via animal bodies (milk, cream, butter, egg yolk). Other food products have synthetically produced vitamin A added in order to biologically render fats, such as margarine, similar to the animal fats. The daily requirements of this vitamin amount in the infant to about 1500 International Units. This quantity is contained for instance in one teaspoon of cod liver oil. Colostrum is particularly rich in vitamin A and carotin. 100 cc colostrum contain about 20 to 30 International Units of vitamin A. 100g breast milk contain about 15 to 25 I.U., cow's milk about 30 I.U.

if the cows are fed in the stables or 75 I. U. if they are pastured. Absorption of carotin as well as vitamin A from the intestinal tract is dependent upon a normal function of fat digestion. In case the fat digestion is disturbed, such as in celiac disease or cystic degeneration of the pancreas, absorption of vitamin A is also affected.

Apart from disturbances of fat absorption hypo—or avitaminosis A also develops when the vitamin A carriers, the animal fats, are completely or almost completely lacking in the diet. The pure form of this nutritional disturbance, the dystrophia alipogenetica, develops only in case of severe lack of fat in the diet of the infant, for instance in serious offenses against the principles of infant nutrition and only rarely from other causes. In order to meet the requirements of the antikeratomalacia vitamin the small quantities of fat contained in half milk or even in buttermilk are sufficient. Only if an infant is fed over a longer period of time with completely defattened milk or fed exclusively with a flour diet (see damages due to excessive flour intake) the fat content is reduced to a degree where a nutritional disturbance develops. As with all other nutritional disturbances the occurrence of the disease is the more likely the younger the infant and the longer the faulty nutrition had been given. The deficient intake of vitamin A alone probably does not suffice to bring about the development of pathological symptoms. As in all cases of avitaminosis, the most important factors are intercurrent infections. Characteristic pathological signs develop almost always only when the child suffers from an infection. During every infection the metabolism and consumption of all vitamins, and among them the vitamin A, is probably increased.

Vitamin A deficiency finally results in Xerophthalmia and keratomalacia. Keratomalacia is, however, not the only symptom appearing as a consequence of deficient vitamin A intake. Even before the development of pathological changes in the eyes uncharacteristic disturbances in the development become noticeable. The skin of the children becomes greyish and dry. A severe dystrophy is observed, which might be due primarily to the excessive reduction of fat deposits and the extremely great dysergia. Only about six weeks after the appearance of these warning symptoms eye changes occur at the occasion of a new severe infection which lend the dystrophy a specific appearance. Dry spots develop on the edge of the cornea in the area between the eyelids, on the conjunctiva bulbi, so-called *Bitot's* spots which spread rapidly so that the injected conjunctiva appears within a short time cloudy and wrinkled. Simultaneously the cornea assumes a clouded appearance, loses its luster and looks opaque or punctuated. This infiltration spreads on the surface and in depth and can finally lead to perforation of the softened cornea.

Although "dysergia," that is, the tendency to frequent, prolonged infections frequently accompanied by complications, is always one of the symptoms of vitamin A deficiency this lowering of resistance is still no specific symptom of avitaminosis A or hypovitaminosis A. The assumption that vitamin A has a decisive influence on the resistance of the child is today rejected. Dysergia is an indirect result of dystrophy which in turn, as in most cases of avitaminosis, is a result of a lack of appetite which occurs in patients with a deficient intake of one of the important vitamins.

Although it is certain that keratomalacia can be traced back to a deficiency of one of the food substances which are bound to fat, only a few children react to a similar degree of deficiency with pathological symptoms. A strong individual disposition is necessary for acquiring keratomalacia. Factors which promote the appearance of the disease are for instance lack of viability and prematurity of the child. The promoting influence of an infection has already been mentioned earlier. A number of infections such as measles or severe consumptive tuberculosis seem to favor particularly the development of keratomalacia.

Treatment of keratomalacia requires supply of the missing substance. Vitamin A containing fat such as milk fat, cod liver oil, etc., must be given as quickly as possible, or vitamin A directly in adequate quantities (5000–10,000 I.U. daily). If changes in the eyes have already occurred an oily solution of vitamin A is given, in addition, two to three times daily in form of drops (1 to 2) into the conjunctival sack. Unfortunately this therapy might at times come too late since the specific diagnosis of dystrophy due to vitamin A deficiency can frequently be made only when in a severely damaged organism changes in the eye are observed as last symptom of the disease. It is, however, possible, particularly in older infants, to cure the disease by rapid and ample vitamin A and fat intake. In these cases the diseased cornea recuperates surprisingly quickly and even severe damages disappear within a short time. But even after curing the local eye damage and after rapid cure of the dystrophy the resistance of the organism against infections remains lowered for many months.

Hypervitaminosis A

Oversupply of vitamin A is accompanied by serious pathological symptoms. Arctic explorers who lived for a long period of time only on the meat of polar bears had known for many years of the "venomousness" of polar bears. The development of damages due to oversupply sets vitamin A apart from other vitamins such as vitamin B or C which, if ingested in excess, never leads to pathological symptoms. The amounts of vitamin C which exceed the requirements are immediately eliminated in the urine.

Oversupply of vitamin A causes typical symptoms only after several weeks or months. Thus damages due to excessive vitamin A intake have so far never been described in children under one year of age. Children develop a lack of appetite and lose weight. Simultaneously subfebrile temperatures appear. The skin itches and assumes a yellowish hue. Hair becomes thin and sparse. The bones are painful. Light edema is observed, also in the face. The liver is enlarged. The children are nervous, irritable and tire easily. The blood serum shows high values for vitamin A and carotin. At the same time the serum lipoids are increased. The alkaline phosphatase in the blood increases to 35 Bodansky units and more. Periosteal thickenings appear in the diaphyses of the bones, particularly ulna, clavicula, femur and tibia, resulting in pictures resembling those of cortical hyperostosis (osteoid-osteoma). The pathological symptoms disappear a short time (10 to 15 days) after discontinuation of vitamin A medication.

The result of oversupply of vitamin A is a defective absorption or rather an accumulation of vitamin A in the liver. The liver becomes damaged, which is manifested by its enlargement. Because of this danger the determination of the vitamin A dosage should be made with caution. In the normal diet of the infant containing milk fat, egg yolk, vegetables, etc. the amounts of vitamin A content suffice for the normal requirements of the infant. Vitamin A should not be prescribed automatically (for instance in combination with vitamin D). It should be prescribed only for patients who show already signs of hypovitaminosis or avitaminosis A (see p. 262) or it should be saved for periods and in diseases (e.g., severe measles) which are known to be connected with a steep rise in vitamin A requirements. We have never been able to convince ourselves of the advantage of prolonged vitamin A medication in e.g., chronic blepharitis and conjunctivitis. An improvement in the susceptibility of a child to illnesses cannot be expected after prescribing vitamin A.

VITAMIN B COMPLEX

Vitamin B was the first substance which was recognized and described as a vitamin. The thermolabile substance which, if lacking, causes beri-beri and the presence of which cures the specific form of polyneuritis was, however, only part of a complex of vitamins which even today is perhaps only partly known. The vitamin B complex includes the following substances of practical importance:

Thiamin (Aneurin, Vitamin B1)
Riboflavin
Vitamin B6
Vitamin B12
Folic Acid
Niacin (Nicotinic Acid)
Biotin

In addition the further components of the vitamin B complex include:

Pyridoxine
Pantothenic Acid
Para-amino-benzoic Acid
Inositol

This last group is no doubt of biological significance. So far as we know today, however, its absence does not result in any known pathological symptoms.

All parts of the vitamin B complex are contained in all types of whole milk, human breast milk, cow's milk, in egg yolk, yeast, fish, in many plants (e.g., spinach), in the hulls of grain (e.g., in the silvery skin of the rice kernel).

Vitamin B_1 deficiency (Thiamin, Aneurin) in the diet produces in the infant, directly or indirectly, that is, in case of vitamin deficiency in the diet of the nursing mother, symptoms of beri beri, of polyneuritis. The patellar reflexes disappear. The patients suffer from constipation as a result of atony of the intestinal wall. Tachycardia and dilatation of the heart develop. Finally a state of dystrophy is observed, either accompanied by edema or of a "dry" type. The daily vitamin B_1 requirements of the infant amount to 200 to 400 γ depending on age. Breast milk and cow's milk contain 200 γ per liter. This quantity is not sufficient for the older infant who then depends on the additional intake of vitamin B_1 carriers such as vegetables, egg yolk, etc.

Vitamin B_2 (lactoflavin or riboflavin) deficiency causes the development of rhagades in the corners of the mouth, similar to the perlèches which, however, can appear also for other reasons. The mucosa of mouth and lips becomes strikingly red, shiny, smooth and dry. Eczema-like changes occur on the skin, the hair becomes dry and sparse. The conjunctiva is reddened and injected. Vitamin B_2 is contained in yeast, egg yolk, in vegetables and in milk where it produces the greenish-yellow color of the whey.

Niacin (*Nicotinic acid, acidum nicotinicum*) is the substance which prevents the development of pellagra. A lack of the substance in one-sided nutrition, for instance on a diet of maize exclusively or on a diet which lacks milk, meat, liver, egg—that is, substances which are rich in this vitamin—causes symptoms of pellagra; this manifests itself in the severely dystrophic infant by the appearance of rough, scaling brownish to blackish skin, particularly over knee and elbow joints and on portions of the skin exposed to light. The tongue of the patient becomes smooth and swollen. Ulcerative stomatitis and diarrhea develop. The patellar reflexes are increased. Psychic changes in form of

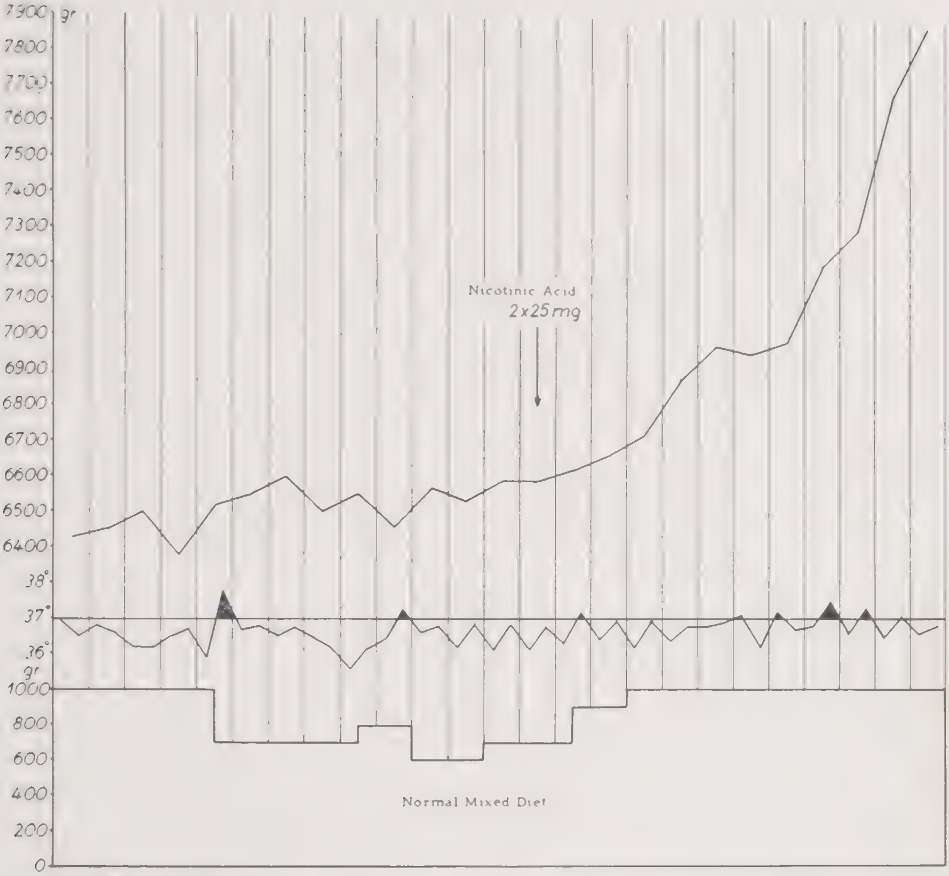

Fig. 30. Infant M.J., 12 months old. Severe dystrophy and symptoms of pellagra. Typical skin changes on elbows, knees, face; hyperpigmentation, irritability, restlessness. No diarrhea. No fever. Disappearance of all pathological symptoms, return of appetite and pleasant mood following medication of nicotinic acid.

worried restlessness which in the older child can develop into a true psychosis can already be observed in the infant suffering from a lack of nicotinic acid. The normal requirements of nicotinic acid are 10 to 20 mg in the infant. Hypovitaminosis resulting from a deficiency of this vitamin can be cured by administering daily quantities of 50 to 100 mg.

Folic acid. The term folic acid or "leaf acid" was chosen because of the abundant presence of this vitamin in green leaves such as spinach. This vitamin stimulates blood regeneration in the bone marrow. Erythropoiesis and leukopoiesis are increased.

This component of the vitamin B complex is therefore indicated in the treatment of alimentary, macrocytic anemias (e.g., goat's milk anemia, see later). Furthermore folic acid has a curative effect on disturbances in absorptive processes in the intestinal tract, such as are characteristic for celiac disease. In a few patients suffering from celiac disease it is possible to improve the diarrhea, to increase food absorption and to raise the general condition of the patient by medication of folic acid, in addition to the dietary treatment (see chapter on celiac disease).

Vitamin B_{12} is probably the anti-pernicious anemia principle contained in the liver extracts which are used in the treatment of pernicious anemia. The effect of vitamin B_{12} medication manifests itself in a sharp rise in reticulocytes in the blood. At the same time there is an improvement in the neurological symptoms of pernicious anemia.

> So-called megaloblastic anemia of young infants can be cured by giving vitamin B_{12}. In this type of anemia megaloblasts and erythroblasts are found in the bone marrow in large quantities. The findings include a leukopenia or neutropenia, reduced number of platelets with tendency to minor hemorrhages in the skin. Without treatment a large number of infants suffering from this form of anemia will die. The cause of the anemia can be inadequate, vitamin B_{12} deficient diet of the mother during pregnancy In some infants a one-sided milk diet, particularly with goat's milk, leads to similar symptoms. In a number of children medication with folic acid seems to be more effective than B_{12}.

Vitamin B_{12} is contained in liver, fish, carrots, spinach, whole grain rice and eggs. Vitamin B_{12}, however, is more readily destroyed during cooking than most of the other vitamins of the B complex. A milk diet, with the addition of only boiled egg yolk and boiled vegetables, therefore does not cover the vitamin B_{12} requirements in predisposed children.

Biotin (H-factor) is today used in the treatment of seborrheic dermatitis in young infants (see p. 501). Even severe cases of seborrheic dermatitis occurring in the first three months of life can be cured within two to three weeks by daily administration of 5 to 10 mg Biotin. In clinically similar appearing conditions of older infants or larger children Biotin does not show the same successful results.

The majority of deficiency diseases which develop due to lack or insufficient intake of one or more of the vital factors of the B complex are accompanied by dystrophy. In these cases the dystrophic conditions are probably not a direct result of the deficiency of one or the other of the B vitamins. These dystrophies develop rather as a result of lack of appetite and displeasure in eating which occurs in any vitamin B deficiency. The secondary dystrophy disappears as soon as the appetite of the children returns with adequate intake of vitamin B. On the basis of these experiences it is today customary in many places to prescribe the entire vitamin B complex for children who fail to develop or who eat badly, even if there are no definite symptoms of a hypovitaminosis B. This routine medication will be successful only in a small part of the patients in whom the dystrophy was, one might say, accidentally the result of a deficiency of one or the other of the B vitamins. As long as there are no definite symptoms such as develop with a lack of one of the components of the B complex this medication will in most patients be unsuccessful. All patients who for some reason (e.g., diarrhea) must avoid for a longer period of time the intake of all vitamin B containing substances must always receive an enteral or parenteral supply, particularly of riboflavin, perhaps also nicotinic acid, in order to prevent the development of hypovitaminosis.

VITAMIN C (SCURVY, MORBUS BARLOW)

Scurvy in children occurs as a result of vitamin C deficiency. Vitamin C is contained in fresh fruit juices, in fresh vegetables, in milk, meat, etc. The identity of "scurvy" of the adult, a disease which has been known for centuries, and the disease as it occurs in infants and small children, which differs in various respects, has been recognized only toward the end of the 19th century (1883). While the symptoms were formerly described by *Möller* as acute rickets, only *Barlow* and others succeeded in proving the scorbutic nature of the disease. Today there is no doubt that *Möller-Barlow's* disease is the form of scurvy characteristic for the earliest period of life. The somewhat different manifestations of the disease at this age are caused by the growth processes, by the different composition of the tissues and by the different demands of the organs during infancy and early childhood.

The disease is characterized by three symptoms:

1) Angiodystrophy
2) Dystrophy
3) Dysergia

Angiodystrophy, the capillary fragility, is the characteristic symptom of scurvy which permits differentiation from all other nutritional disturbances. Angiodystrophy causes hemorrhages in the skin, mucosa and in the periost of the bones. Hemorrhages in the skin occur during infancy more in the face while the lower extremities—in contrast to the adult—are much less affected. Hemorrhages occur most frequently around the eyes, in the eyelids, on the cheeks, in the auricles which are sensitive in the infant due to the absence of cartilage, and in the folds of the neck. The hemorrhages appear in form of punctate or more spread out, reddish to reddish-blue discolorations of the skin which later turn blue and green. Some of the hemorrhages disappear rapidly without undergoing these changes. Hemorrhages of the mucosa occur most frequently in the mouth and in the urinary passages. Bleeding of the gums which is characteristic for scurvy in the adult occurs in infants, with few exceptions, only after the teeth have erupted. The bleeding is usually preceded by a period during which the gums are reddened, swollen and loosened. In the area of the hemorrhages in the non-resisting mucosa we find not infrequently, particularly in small children, secondary infections which simulate the symptoms of stomatitis aphthosa or ulcerosa. Apart from the gums, bleeding occurs in the oral mucosa on the hard and soft palate, and in the mucosa of the cheek, usually in the area of the jaw occlusion and the musculature of the tongue.

Hemorrhages in the urinary passages appear in form of a macroscopically visible hematuria or as only microscopically discernible erythrocyturia minima in 90% of all cases of scurvy. The intestinal mucosa can also be affected by the hemorrhages. More severe hemorrhages, accompanied by diarrhea which occurs not infrequently in children suffering from scurvy, can simulate dysentery.

Hemorrhages under the periost cause painful enlargements of the bones which occur almost exclusively in the areas of the epiphyses of the long bones. They also appear under the periost of flat bones of the skull, of the orbita or at the chondrocostal junction of the ribs. In all these subperiosteal hemorrhages there is always a danger of secondary infections. Hemorrhages under the periost are, however, only part of the changes which take place in the skeleton in case of scurvy. The growth area of the bones undergoes far-reaching changes which originate in the bone marrow which is replaced by connective tissue, poor in blood vessels and rich in young connective tissue cells. The absorption of temporarily calcified parts of the epiphyseal line continues undisturbed while new formation of bone substance does not take place due to defective activity of the osteoblasts. The result is a resorption of spongiosa and cortex of the bone. The bones become thin and brittle and a minor mechanical trauma suffices to bring about fractures and dislocations in the most seriously diseased area, the growth zone. Dislocations of this sort can almost always be observed on the costochondral junction with the development of a step-like discontinuity of the ribs which has been described as scorbutic rosary. In this manner the orderly structure of the epiphyseal line develops into the area of destruction (Trümmerfeldzone), a confused accumulation of calcium and bone trabeculae, of marrow rich in connective tissue and of fresh and older blood. These disturbances in the bone structure together with the subperiosteal hemorrhages and edema of periost, cartilage, musculature and skin result in the painful swellings which are characteristic for scurvy. Pains which are observed every time the child is touched, when bathing or when changing the diapers, are not infrequently the earliest symptoms of the disease.

Dystrophy and dysergia are non-characteristic symptoms which occur with any type of nutritional disturbance. Dystrophy with reduced weight gain or no weight gain at all, frequently also with interruption in growth in length is almost always present. This lack of development is again the result of a lack of appetite with which all children suffering from scurvy are affected. In part this lack of appetite is also a result of psychological changes which manifest themselves in form of irritability and negativism toward the surroundings in all scorbutic children. Bad humor, crying and yelling, with never a smile, are characteristic for the psychological attitude of the scorbutic patient.

Apart from dystrophy, dysergia is present as a non-specific symptom in most cases of scurvy. Dysergia is the changed attitude of the organism in fighting off infections. The ability for normal resistance against infections is lost. The weakness in bacterial resistance manifests itself in the above mentioned tendency of hematomas to become infected. But also any other infection with which a child suffering from scurvy is afflicted develops into a prolonged illness, accompanied by high temperatures, which can spread without encountering resistance. The increased vitamin C requirements with every kind of infection promote in turn the development of specific and non-specific scorbutic symptoms. Dysergia is also of decisive significance for the prognosis of scurvy. Children suffering from scurvy who receive no treatment or very late treatment do not die of scurvy itself but of an infection.

The symptoms of scurvy never develop in the course of a few days. This last stage is always preceded by a longer or shorter period of time during which less striking specific and non-specific symptoms, which at first are difficult to interpret, point to the threatening danger (subclinical scurvy, abortive scurvy). Depending on the amount of deficiency in vitamin C intake this period lasts from four to eight weeks. Dystrophy and dysergia precede the disease. Among the non-specific symptoms it is again dystrophy which appears first, together with a striking lack of appetite. Almost simultaneously the resistance against infections becomes lowered. Temperature elevations do not yet occur more frequently than in well developing children of the same age. However, instead of temperature elevations lasting a few days the fever periods extend over many days, even weeks. In comparing several consecutive infections one can observe that every new infection lasts longer, is accompanied by higher temperatures and is more subject to complications than the preceding one.

In addition to dystrophy and dysergia we then observe the first signs of hemorrhagic diathesis. A few erythrocytes appear in the urine, and a more or less dense dissemination of petechiae develop on the face, usually around the eyes and the mouth, which disappear again after a short time. These minimal petechiae occasionally occur already at a time when the dystrophy is still little developed. The justified suspicion that these are hemorrhages resulting from a disease connected with a bleeding tendency is excluded by findings of normal values for blood platelets, coagulation time, bleeding time, and by findings of a normal bone marrow. The close connection between these hemorrhages and the occurrence of elevated temperatures is, however, remarkable. These minute hemorrhages appear a few days or hours before the signs of an infection or even only at the onset of fever or within a few hours afterward. Internal hemorrhagic pachymeningitis also develops on the basis of scorbutic bleeding tendencies and must be included among the symptoms of scurvy in infants. The brittleness of the vascular system can be proved in the preclinical stage of scurvy by the positive result of the *Rumpel-Leede* test. This symptom is, however, not pathognomonic for scurvy.

For confirmation of the diagnosis roentgenographic examination, particularly of the long bones, is of great value. Already at an early stage the area of destruction (Trümmerfeldzone) in the atrophic bone appears as a broad, dense, irregularly outlined shadow. This picture, which remains visible for many months after scurvy is cured, is not always a sufficient indication for the presence of scurvy. It is supplemented by a change in the bone centers. The roentgenogram shows a circular shadow around each bone center which thus assumes the appearance of a "balloon" (ring). The cause for these circular shadows are changes in the periphery of the bone centers which are analogous to those of the zone of destruction.

The diagnosis of scurvy can present difficulties in the noncharacteristic preclinical stage. But even the imposing symptoms of fully developed scurvy are not infrequently misjudged. Errors occur particularly in those cases of scurvy where one particular symptom such as swelling of the bones or the pseudo-paralysis is particularly outstanding or where hemorrhages in the kidney dominate the clinical picture. Swelling in the areas of individual bones of joints might lead to an erroneous diagnosis of fractures which are treated unnecessarily with bandages, or to a diagnosis of osteomyelitis or tumors which has been known to result in superfluous, disfiguring operations. The pseudo-paralysis which is caused by the painfulness of every movement can simulate a neuro-paralysis or can be confused with *Parrot's* syphilitic pseudo-paralysis. The presence of all reflexes and the considerable tenderness of other limbs which are not affected by the paralysis speak against nervous paralysis. The age of the child speaks against a syphilitic paralysis. Syphilitic pseudo-paralysis occurs in the first three months, scorbutic pseudo-paralysis not before the end of the first half year of life. The location of the paralysis also permits a certain degree of differentiation. Syphilitic paralysis occurs more frequently in the upper extremities. The most severe bone changes in scurvy usually take place in the lower extremities. The hemorrhages in kidney and intestinal tract might also lead to a wrong diagnosis. In cases of hematuria one

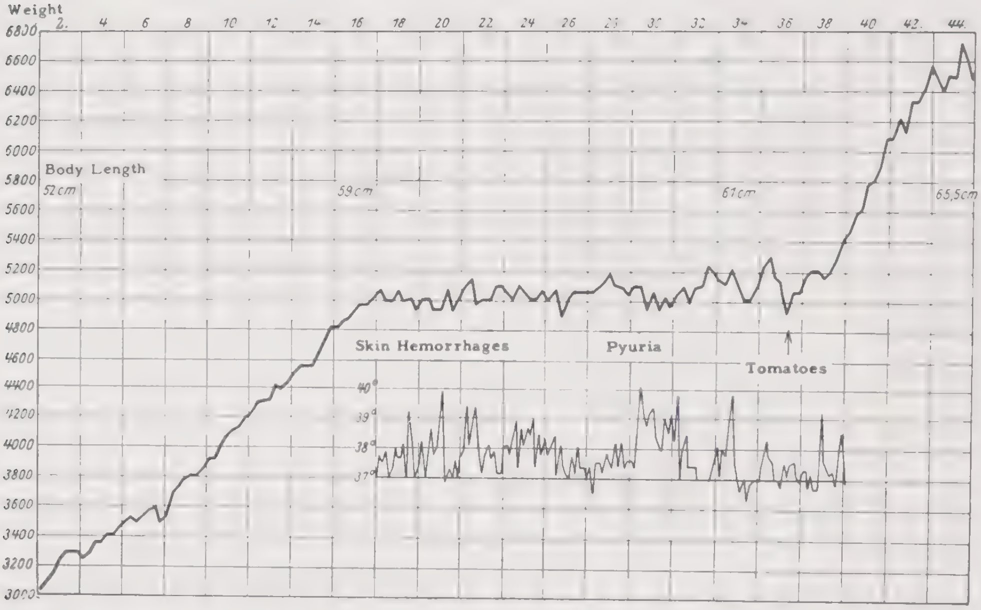

Fig. 31. Rapid cure of scurvy by addition of fruit juices to the diet. Disappearance of dystrophy and dysergia and cessation of hemorrhages.

might think of hemorrhagic nephritis or hemorrhage due to stones. Intestinal hemorrhages have occasionally led to a diagnosis of dysentery, even of ileus. Only a careful examination of the child which reveals other symptoms of scurvy can protect the physician from such errors. This careful searching is particularly necessary in the abortive forms of the disease.

The cause of scurvy is a vitamin C deficiency in the diet. Vitamin C could be chemically identified as ascorbic acid and produced synthetically. Ascorbic acid is available in form of tablets or as solution for intramuscular or intravenous injection. Ascorbic acid loses its specific qualities upon contact with oxygen or with certain metals (e.g., copper). This sensitivity is important for the boiling of milk, for the preparation of powdered milk, of canned fruit juices and other foodstuffs where the vitamin C content is to be preserved. Pasteurizing of the milk destroys part of the vitamin C. Repeated boiling of milk destroys the anti-scorbutic factor.

A moderate degree of vitamin deficiency does not in all children lead to scurvy. On the other hand there are children where it is impossible to prevent scurvy despite a certain amount of vitamin intake. Individual disposition is of particular importance in scurvy which has also been confirmed in animal experiments. There are, after all, a number of infants whose development will be undisturbed even without addition of vitamin containing substances. The vitamin requirements of the growing infant are low, certainly much lower than those of certain animal organisms such as the guinea-pig.

Scurvy of the infant is a disease of winter and spring. More frequent febrile grippal infections, lack of fresh vegetables, difficulties in obtaining sufficient quantities of adequate vitamin carriers (fresh fruit juices) and a lowering of the vitamin content of cow's milk during the dark season favor the development of scurvy. Scurvy can always be cured by giving adequate quantities of vitamin C containing foodstuffs. By suitable prophylactic supply of vitamin carriers scurvy can always be prevented. It must be remembered, however, that the amounts of vitamin intake must be adapted to the increased requirements in case of infection or nutritional disturbances.

In order to prevent the consequences of a vitamin C deficiency an excessive rather than an insufficient vitamin supply might be indicated in the practice of nutrition. Damages caused by excessive vitamin C intake (hypervitaminosis C) are not known. The excess amounts of vitamin C are not stored up but are eliminated immediately through the kidneys.

The vitamin C requirements of the infant amount to about 50 to 80 mg per day. Breast milk contains variable amounts of vitamin C depending on the vitamin content in the diet of the nursing mother. With a normal mixed diet of the mother an average vitamin content of 30 to 50 mg per 1000 cc of milk can be assumed. Cow's milk contains an average of 10 to 30 mg vitamin C per liter, depending on the type of fodder of the cows (green fodder, pasture, etc.). Scurvy develops when the vitamin C requirements are not covered. Vitamins do not act in the metabolism in the capacity of a catalyzer. Insufficient quantities of vitamin C containing substances, given by teaspoon or even by drops, cannot prevent scurvy.

Various types of foodstuffs differ in their vitamin content and even the same type of fruit might show variations in vitamin content depending on sun irradiation, fertilization and degree of ripeness. Since it is not possible in practice to examine the vitamin contents of the available milk, fruit and vegetables, it is advisable to use types of fruit known to be rich in vitamins. Among the fruit juices the following can be utilized in summer: cherry juice, fresh raspberry juice, strawberry juice, grape juice and tomato juice. During the winter months tomatoes, oranges, lemons, grapefruit are recommended as long as the fruit is not overripe or withered. All other juices such as carrot juice or pressed juices from vegetables which are said to have an antiscorbutic effect are less effective or not effective at all. But even certain commercially and technically adequately prepared and canned fruit preserves contain sufficient amounts of vitamin C. Scraped apples or pears and even bananas are relatively poor in vitamin C. The quantities of fruit juices necessary for effective prophylaxis should amount to not less than 30g (2 tablespoons) per day. These quantities, however, apply only to the healthy and well developing child. The quantity must be increased in case infections or other diseases occur, to amounts of about 50 to 100g per day. Intake of fresh fruit juices in excess of these quantities is of no value since vitamin C cannot be stored in the organism.

The time at which prophylactic vitamin intake should be started in the infant is determined by the observation that scurvy never develops before the fourth to fifth month of life and that in case of vitamin C deficiency the first symptoms do not appear until two to three months later. Therefore, the addition of vitamin containing juices is not absolutely necessary during the first two months of life. In the third month of life at the latest, however, addition of fresh fruit juices should be started. This supplementation is recommended not only for bottle fed children but also for breast fed infants. After the period of infancy when the intensity of growth processes and of development is reduced the vitamin requirements also seem to decrease gradually so that smaller quantities of fruit juices are sufficient to cover the requirements.

In hardly any other disease can severe illness be cured as rapidly as in adequately treated scurvy. A child which cried at every touch and appeared almost paralyzed moves about in his bed within a short time freely and without pains. Hemorrhages disappear rapidly. Kidney and intestinal hemorrhages cease. The temperature elevations which almost regularly accompany the onset of scurvy return to normal within a surprisingly short time. Children who suffered from constantly recurring infections during the pre-clinical stage of scurvy now develop without any further disturbances. The changes in the bones also heal. New bone formations appear in the areas of subperiosteal hemorrhages. The strength of the bones increases. Only in cases where displacement had already occurred in the continuity of the bones in the areas of destruction or in the costochondral junction these changes remain visible for a long time as left-over remnants of the disease. With the disappearance of acute pathological symptoms the dystrophy too, which had existed for many weeks and months, regresses. A new phase of undisturbed development begins for the child.

For treatment of scurvy quantities of about 100g fruit juice per day are adequate. The intake of these large quantities should be continued until the development of the child is satisfactory. Only when there is no longer a tendency for the reappearance of scorbutic symptoms, when the scorbutic diathesis has disappeared, the quantities of vitamin carrying substances can be reduced to the smaller amounts as recommended for the prophylaxis of scurvy.

In those cases of the disease which show only dystrophy with abortive symptoms of scurvy the same good results can be obtained by this treatment. This applies particularly to dystrophic children who suffer from recurrent infections around the sixth month of life and who betray the presence of scurvy only by occasional, minimal hemorrhages in the skin and the mucosa (erythrocyturia minima). Here, too, the dystrophy can be cured by the supply or the increase of the previously given quantities of vitamin C. Diarrhea is no contra-indication for prescribing fruit juices. Moderate quantities of fresh fruit juices are well tolerated in cases of diarrhea, particularly when the cause of the diarrhea is connected with a vitamin C deficiency.

The pathogenesis of scurvy, that is, the manner by which vitamin C deficiency leads to pathological symptoms, is not yet clear. It is possible that vitamin C deficiency leads to defects in the intercellular connective substances in the capillary walls and connective tissues. In the vascular system they lead to greater permeability of the vascular walls and thus to hemorrhages, transudates and edema.

VITAMIN D

Rickets

Rickets is a disease of progressing civilization. During antiquity it was known already in large cities as a children's disease and during the seventeenth century it spread rapidly in England with the development of industrialization of the country. With the growth of cities and the movement away from the natural mode of living the number of children suffering from rickets has increased considerably. Depending on social conditions, on adequate infant care and suitable application of prophylactic and therapeutic methods existing today, a larger or smaller number of infants and small children are afflicted by the disease. In contrast to tetany, rickets does not spare breast fed children.

> The interpretation of the character of rickets has undergone changes in the course of the centuries. The first reports of the disease place it among the dyscrasias. Under the influence of the newly developed pathological anatomy it appears as a localized disease of the skeletal system. And again, under the influence of the study of bacteriology which at times dominated the entire field of medicine, it is placed among the infectious diseases although not with the consent of all authors.

At present rickets is considered a disease of the entire organism, with the most striking clinical changes occurring in the skeletal system, and caused by an insufficient intake of vitamin D. The great majority of all rachitic diseases heals spontaneously around the third to fourth year of life, although the changes which occurred as a result of the disease frequently remain throughout later life.

The manifold changes on the skeleton occurring in rickets do not appear in a haphazard manner. The location of rachitic signs varies depending on the age of the child so that rickets might be fully developed in certain parts of the skeleton while other sections are at least clinically free of changes. In other words, while in one place the pathological process of the disease has already come to a standstill or has healed, the disease only begins to develop in another bone. These changes in the location of the disease follow a certain pattern. The rachitic bone process affects those bones the strongest which at the moment are in the most intensive period of growth, such as the skull in the first quarter, the thorax in the second quarter of life and the extremities in later months. Accordingly, the rachitic bone process descends beginning with the skull, over the thorax to the extremities, so that it has been described as the wandering of rickets. The temporary rapid growth of certain bones results in particularly high calcium requirements in certain parts of the skeleton. The result of this sequence of rachitic symptoms whereby healing processes occur simultaneously with newly developing changes and with quiet areas of previously healed processes produces the variable picture of rickets.

Rickets of the skull, craniotabes, is the earliest symptom of the disease. It appears during the second to third month of life in form of a softness of the occipital bone. In prematures this softness occurs still earlier. Rachitic craniotabes should be differentiated from the not infrequently occurring softness of the skull bones, the so-called congenital soft skull which develops in utero and is already present at birth and which is caused by mechanical pressure of the pelvic bones of the mother on the skull. The seat of the symptoms in congenital soft skull, the parietal bone, permits differentiation from craniotabes which occurs only in the occipital region. Congenital softness of the skull is centered on or around the sutures while craniotabes is located more in the center of the os occipitale. Congenital soft skull is not accompanied by changes in bone structure and changes in the blood chemistry which are characteristic for rickets.

Rachitic craniotabes occurs in many breast fed infants and not only in artificially fed children. Temporary softness of the skull of varying degree and varying duration occurs also in well developing children, at times even despite prophylactic treatment. However, more severe cases of craniotabes of longer duration occur only in children living under unfavorable conditions which promote the development of rickets. The severest degrees of craniotabes are observed in cases where the growth of the skull bones is particularly rapid. This happens in children with hydrocephalus and in prematures where the relatively intensive growth of the brain in turn causes a particularly rapid growth of the skull. In these children the entire occipital region changes at an early age into an almost paper-thin layer which yields to pressure of the hand by cracking like a celluloid ball. On the other hand craniotabes does not occur in children with reduced skull growth, e.g., in case of microcephalus. Craniotabes has the tendency to heal spontaneously. In the full term child it is hardly ever observed beyond the first half year of life, even if severe rachitic changes are found in other parts of the skeleton.

After about the third to fourth month of life rickets of the thorax develops next to craniotabes. It appears as "rachitic rosary" in form of a row of bead-like swellings on the chondrocostal junction. It is not possible to determine the time of onset of rickets of the thorax. The appearance of the symptom also does not permit a conclusion as to whether it is new or already healed rickets of the torax. The changes remain visible for months and years, even after all the other criteria for the cure of rickets have been fulfilled. The florid rachitic process on the chondrocostal junction is soon followed by an abnormal softness of the entire thorax including the clavicles. The results of this softening of the bones are deformities of the thorax. The normal curvature of the thorax disappears. More pronounced flattening and indentation from the side, producing the pigeon breast, occur only in the later months of infancy. A so-called funnel chest might develop. The parts of the thorax which lie below the insertion of the diaphragm are turned outward. In this manner the so-called *Harrison*'s groove develops which follows approximately the costal insertions of the diaphragm. These severe deformities of the thorax remain in existence until later childhood, they even determine frequently the form of the thorax of the adult. All these changes, such as funnel chest, *Harrison*'s groove, etc., are not always caused by rickets. They are just as frequently congenital or inherited forms of development of the thorax.

Due to the softness of the thorax the decalcified ribs are pressed inward during inspiration so that the lungs cannot expand normally. The lungs of the rachitic child therefore remain small. A congestion develops in the badly ventilated lungs leading to congestive bronchitis, atelectasis and a tendency to pneumonia. The poor development of lungs and thorax and its consequences endanger the life of the rachitic child. A child does not die of rickets itself. The greater mortality of severely rachitic infants is caused by atelectasis, broncho-pneumonia, bronchiolitis which develop independently or as complications of other infectious diseases, particularly measles and pertussis.

Rachitic changes in the extremities do not become evident until the second half of the first year of life. They appear as enlargements of the epiphyses in form of rachitic enlargements to both sides of the joint. The softening of the bone shafts leads to deformities of the long bones. Static pressure is not necessary for these processes and therefore deformities occur also in the arms.

The pull of the musculature whereby the force of the flexors always exceeds that of the extensors suffices to bring about severe concave deformities towards the inside even in children who have never stood or sat up. Weight-bearing on the bones will further promote the deformity. Thus the bones of a child suffering from active rickets of the extremities appear plump and enlarged and broad in the epiphyses. These forms of rickets occur more frequently in the young child than in the infant during the first year of life. Signs of active rickets are a saddle-formed indentation over the malleoli which can be felt by palpating along the length of the bone. This so-called *Marfan*'s sign can be observed only in active rickets.

There is no particular tendency to brittleness in the soft rachitic bones. Due to disturbances in the ossification formation of so-called "pearl fingers" is observed on fingers and toes which points toward the involvement of the short bones in the rachitic disease. Rachitic deformities of the spinal column are rare during infancy. The rachitic spinal curvature which appears after the second half of the first year of life is not the result of bone changes but rather the result of a rachitic involvement of muscles and ligaments whose loss of tonus fails to support the osseous spinal column.

There are two reasons why teeth are only little affected in their structure by rickets. The bones which are involved in rachitic disease are part of the mesoderm. The enamel originates from the ectoderm. The degree of changes in the teeth and severity of rachitic processes thus do not run parallel. Furthermore at the time of onset of rickets the calcification of most tooth buds is already completed, even of those teeth which are still unerupted so that the signs of rachitic disease can hardly affect the hard enamel. Thus we do not find more carious or defective milk teeth in even severely rachitic children than in non-rachitic children with the same social background. Onset of teething also seems to depend more on familial and hereditary factors than on the presence or absence of rickets. If rachitic children occasionally develop serious defects on the teeth these changes are a result of other unfavorable living conditions (inadequate housing, faulty nutrition, frequent infections) under which these children are raised.

Apart from bones rickets affects the entire musculature, the ligaments and the nervous system. The musculature of the rachitic child is withered, slack and of poor tonus. This hypotonia is not caused by anatomical changes in the muscles, it is caused by disturbances in the nervous supply which dominates the tonus of the musculature. Disturbances in the vegetative system and in the development of the nervous centers on which appearance and maturation of static functions depend, result in delayed development of sitting, standing and walking. The involvement of the musculature in rickets is independent of the severity of bone disease. Children with most severe active rickets and most pronounced deformities of the extremities need not always be backward in their muscular development. A further result of inadequate muscle tonus is the enlarged abdomen of the rachitic patient which is caused by the slack tonus of the abdominal musculature and by loss of tonus of the smooth intestinal musculature conditioned by disturbances of the nervous supply.

Anemic conditions of varying degrees are observed in rachitic children. They have at times been brought into direct etiological connection with rickets. Examination of the bone marrow did not reveal anything which would speak for an involvement of the hematopoiesis in rickets. Even in most severe cases of rickets no changes in the blood may occur. Since faulty nutrition and recurrent infections are frequently observed in the etiology of rickets the anemic conditions occurring in the rachitic infant should be regarded as sequellae to alimentary or infectious factors. The enlargement of the spleen which is observed in a number of rachitic infants is also a symptom of anemia rather than of rickets.

The severe sweating which occurs in some of the rachitic children and which develops even without any physical effort, even during sleep, particularly on forehead and occipital region, is the result of damage of the sweat glands due to disturbances of the vegetative system. It has not yet been determined whether this symptom is to be attributed to rickets or to infantile tetany. In children with strong tendencies to profuse sweating history or symptoms usually reveal signs of latent or manifest tentany.

The central nervous system is not spared by rickets. The psychological development is disturbed, brought to a standstill or even shows retrogression. Previously acquired abilities are lost again. Static functions which had been present are no longer practiced. New acquisitions of mental functions do not take place or take place with considerable delays. This retardation in the development of the nervous system occurs by no means in all rachitic children. Here, too, the degree of bone involvement does not give any indication as to the degree of expected psychological disturbances. In cases where disturbances occur in the areas of intellect and emotions they affect the ability of perception. Simple tasks which are used to test the intelligence of older infants can no longer be solved. All reactions are slowed down. The motor actions also are affected.

Occasionally conditions develop which resemble catatonia. Loss of the urge to move about and of natural agility is in many cases a lower degree of motoric disturbance caused by rickets. The intellect itself is affected to varying degrees in the individual rachitic children. Some of them are content and in their behaviour hardly differ from the normal child. Other children develop disturbances which in the most serious stages resemble true oligophrenia. The term "dementia rachitica" has been used. The rachitic child can become apathetic or rejecting, negativistic. Stereotypies develop which include spasmus nutans and similar mannerisms frequently observed in rachitic children. In this condition of emotional rejection children frequently respond to any rapprochement with crying. This creates the impression that any contact is painful. Pain, however, is not part of rickets. If the bones are actually painful the child probably suffers from a combination of rickets and scurvy.

The basis of cerebral rickets is perhaps a result of disturbances in the calcium and phosphorus metabolism which also affect the central nervous system. The proven calcium deficiency of the brain leads to swelling of the brain substance and thus to an enlarged head, simulating hydrocephalus, as found in some rachitic children.

The damages and disturbances in the development of motor activities which were mentioned above are only to a small degree results of the disease of bones and muscles. These disturbances are caused mainly centrally due to the fact that the central regulation of motor activities which is necessary for normal statics is damaged. Although the desire and the urge to perform movements is present in many rachitic children they lack the ability to execute the motor activities. Grasping with opposed thumb, which normally occurs around the sixth month of life at the latest, frequently remains in rachitic children even in the middle of the second year of life the grasping or holding on of the young infant, with the thumb in a row with the other fingers and never intentionally placed in opposition. The position of the healthy young infant with strongly flexed thighs is maintained in the resting and sleeping rachitic child even at a time when the healthy child already sleeps with relaxed and straightened limbs.

Tetany was for a long time regarded as rachitic disease of the nervous system (see p. 295). Now, on hand of metabolic tests the principal differences between rickets and tetany have been uncovered. The fact remains, however, that most cases of tetany during infancy occur as sequellae to active rickets.

Pathological anatomical changes and disturbances in metabolism in cases of rickets show that despite abundant calcium intake normal calcification of newly formed tissue does not take place. For this reason the bones become soft and deformed. In place of normal bone structure, containing little fluid and much calcium, we observe the formation of tissues which are rich in fluids and poor in calcium.

On the border between diaphysis and metaphysis the cartilaginous cells normally place themselves in long orderly rows and between them stand parallel columns of cartilaginous bone substance. This cartilaginous structure is periodically and provisionally fortified through deposits of calcium salts in those parts which are situated closest to the epiphysis (osteoid formation). The provisionally calcified cartilaginous columns are surrounded from the vascular bone marrow space by a net of osteoblasts while the originally existing cartilaginous cells are destroyed. The osteoblasts absorb the cartilaginous tissue and to the same extent build in its place the final osteoid tissue. This process, which is repeated constantly during the entire period of growth, is so well synchronized in the healthy individual that we find not only the rows of cartilaginous tissue and the car-

> tilaginous columns exactly parallel but the border within which the transformation from cartilage into permanently calcified bones takes place appears as a sharp line. This line which corresponds to the zone of temporary calcification is also visible in the roentgenogram. The width of the osteoid tissue in the healthy infant is always small since newly formed osteoid tissue is immediately transformed into bone.

The balance between new formation of osteoid and new formation of bone is disturbed in rachitic children. Even provisional calcification fails to take place while rows of cartilaginous cells continue to accumulate in preparation for the process of ossification. Since these initial disturbances do not appear evenly distributed over the entire border between epiphysis and diaphysis the growth zone here loses its normal sharp line of demarcation. In the anatomo-pathological preparation and in the roentgenogram it appears torn and fringed. With prolonged absence of calcification processes we find a gradual accumulation of soft cartilaginous tissue which partly is not even provisionally calcified any longer. The absence of ossification in the diaphysis of the long bones causes here too increased osteoid formation and thus softness of the bones. The onset of healing of rickets can be noted in the re-establishment of a balance between anabolic and catabolic processes and particularly of final calcification. The body forms a new zone of calcification, situated toward the epiphysis, where normal activity of new bone formation takes place in normal rhythm. The quantities of pathological osteoid tissues which were built up by the rachitic process are independently and gradually calcified. The zone in which the bone structure deviates from normal can be determined anatomically and roentgenologically for a long period of time as remnant of the past disease.

As exact criterion of beginning and healing rickets the roentgenogram is today of great value. As a result of the general deficiency of calcium the roentgenogram of rachitic bones shows a thinned out cortex and a large trabecular spongiosa. The osteoid tissue formed in the active stage of rickets does not produce a clear shadow but only a washed out picture so that the entire rotentgenogram appears without much contrast ("dull").

In the severest degrees of so-called osteomalacic forms of rickets the bone can be hardly distinguished from the soft parts on the film. With abundant formation of periosteal osteophytes a marginal line forms parallel to the cortex which because of its low calcium content is also of low intensity (in contrast to the denser double contour in periostitis luetica). If in the absence of symptoms of a fracture a clear zone up to 1 cm width is seen throughout the thickness of the bone it is probably not a real fracture but rather the so-called *Looser's* zone of transformation. Under the influence of muscle pull and static pressure normal bone is changed into bone of a net-like structure and without calcium.

As long as moderate or severe rickets is in the active stage no new bone centers appear in the roentgenogram. If the skeleton is examined at the beginning of the healing process the roentgenogram reveals within a few days bone centers of a size which show that these centers had been present for months in an uncalcified state.

The manifestation of disturbed growth processes in the roentgenogram is the absence of the sharp linear border which normally terminates the bone toward the epiphysis. Instead the shadow of the diaphysis gradually loses itself in form of an indistinct, fringed area into a broad, transparent zone. This border zone shows at times, but not always, a cup-like concavity with elongated borders.

The reasons for the absence of physiological calcification of the preformed osteoid tissue are not known. Disturbances in the absorption of calcium and phosphorus from the intestines, resulting perhaps from a dysfunction of endocrine glands (parathyreoidea ?) might explain these pathological processes. Why the rachitic bone loses its ability for calcium fixation, an ability which sets the healthy bone apart from all other tissues, has so far not been explained.

The attempt to understand the disturbances in the process of calcification on the basis of analyses of bone substances has not brought any results. The relationship of the essential salts contained in the bones, of $Ca : PO_4 : CO_3$ is approximately equal in the normal as well as in the rachitic bone, although the total ash content is somewhat lower in the rachitic bone. Very little is known about changes of the calcium and phosphorus content in other tissues of the rachitic child. The quantity of calcium in the blood serum is not lowered. However, in cases of active rickets we find with great regularity a lowering of the phosphorus content in the blood serum; this is so characteristic for the pathological state of rickets that this finding is today one of the finest indications for the diagnosis of active rickets. Normally the amount of calcium contained in the serum averages 10 mg% (8 to 11 mg%), the amount of phosphorus 4.5 to 5mg%. In the rachitic child the phosphorus of the serum is reduced to 1.5 to 3.5mg%. In the healthy infant the quotient of Ca : P is 2. In case of rickets, when the calcium content is approximately normal and the phosphorus content considerably reduced, the quotient becomes about 3 to 3.5. Accordingly the product of calcium to phosphorus is:

in the normal child $Ca \times P > 40$
in the rachitic child $Ca \times P < 30$

The quantity of alkaline phosphatase in the serum is increased in rachitic children (as in many other diseases of the skeletal system). Phosphatase is a product of the osteoblasts. It is an indicator for the intensity of the work of these cells. Instead of normal values of about 10 Bodansky units in 100 cc serum we find in the rachitic child values of 15 to 60 Bodansky units and more, with the quantity of phosphatase approximately paralleling the severity of the rickets. Return to normal of the amounts of phosphatase in the serum is an even better indicator for the healing of rickets than the determination of phosphorus in the serum or the disappearance of the findings in the roentgenogram.

The cause of rickets is not, as formerly assumed, a primary phosphorus deficiency (see medication of phosphorus containing cod liver oil). Increased intake of phosphorus in the food leads to an increased phosphorus level of the serum and to a healing of rickets only when vitamin D or an equivalent anti-rachitic treatment (such as ultraviolet irradiation) is given simultaneously.

Due to a decrease of the bicarbonate content and the alkali reserve in the blood the rachitic child develops a metabolic aci-

> dosis. This acidosis is the result of a slowing down of the entire metabolic process. Any slowing down of cell oxidation leads to an intermediary accumulation of acid metabolic products. The slowing down of the metabolism in rachitic individuals is caused by the absence of light or by the deficiency of antirachitic vitamin, but above all by a preponderance of products of the endocrine glands which slow down the oxidation process in the organism. The relationship of all these processes to the absence of calcification of the bones has as yet found no satisfactory explanation. Most investigators assume that there is a direct relationship between the hypophosphatemia and the absence of calcium deposits in the bone.

One of the premises for missing calcification is the growth of the bones. Rickets does not develop when the growth processes are delayed or are stopped altogether. The fact that rickets does not occur in cases of myxedema or in non-growing atrophic infants is proof for this assumption. Hunger and malnutrition have a similar effect. These processes disturb the enlargement of the cartilage and thus the growth processes. The fact that active rickets develop only rarely beyond the fourth year of life is to a great extent a result of the gradually decreasing intensity of growth.

In contrast to our lack of knowledge concerning the pathogenic processes, the etiological processes in the development of rickets are better known. Rickets develops in places which are not sufficiently exposed to light and air, under crowded and poor living conditions and in infants receiving inadequate care and nutrition. This rather indefinite theory for the etiology of rickets was propounded until a few years ago. However, lesser degrees of the disease were also observed under good living condition so that in countries with moderate climate traces of the disease could, upon careful examination, be found in almost every infant. It has taken decades until it became possible to separate from the multitude of damages which occur in children living under unfavorable conditions, particularly in large cities, and which were referred to as "domestication", the factor which is decisive for the development of rickets. Two facts are established today: Rickets develops from a lack of sunlight and from certain deficiencies in the diet. Apart from external factors the constitution of the individual is, as in any other disease, of significance for the absence or presence as well as for the degree of severity of rickets. The influence of constitution seems to be more frequently inherited from the mother's side so that we can observe similar pictures of rickets repeating themselves in two or three successive generations. The hereditary factor is of far less practical significance for the development of rickets than the lack of light and the faulty nutrition. Children of families with rickets do, however, require particularly careful prophylaxis.

The significance of lack of light for the development of rickets has been known for a long time. During the dark winter months rickets develops with particular frequency and greater severity. It heals during the sunny months of spring. In mountainous areas rickets occurs much more frequently in the shadowy valleys than on the slopes which are directly exposed to the sunrays. These observations and the resulting practical conclusions for prevention and cure of rickets remained unknown for a great many years. Only about 30 years ago reports appeared about the first therapeutic successes with systematic exposure of rachitic infants to sunshine, which thus proved the significance of light for the curing of rickets. At the same time it was shown that only a certain section of the spectrum, the ultraviolet rays, were of importance for curing rickets. In later years it was shown that the natural sunlight is no less effective than the artificial rays.

The section of the spectrum comprising the effective rays includes rays of a wave length of 230–313$\mu\mu$. This explains why we find no rickets or why rickets heals rapidly in areas along the seashore or in sunny high mountains where ultraviolet rays are particularly abundant. It further explains the absence of rickets in tropical climates, provided that the body of the child is freely exposed to the sun rays. On the other hand the reason for the frequent occurrence of rickets in large cities with their dust and smoke polluted atmosphere which intercepts the larger part of ultraviolet rays becomes understandable.

Apart from the lack of light, nutrition is of significance for the development of rickets. Faulty nutrition was emphasized already by *Glisson* in his first classical description of rickets.

about 300 years ago, together with unfavorable living conditions and unfavorable influences of the constitution. The significance of the nutritional factor was, however, recognized only about 100 years ago when cod liver oil was found to be an effective prophylactic and curative substance for rickets. But even the efficacy of cod liver oil was doubted in the beginning. At first cod liver oil served only as vehicle for the administration of phosphorus which was recommended for the cure of rickets. The effect of cod liver oil itself, which aids in the deposition of calcium in the bones, was recognized only in later years; the effect, however, was observed only if the commercially available cod liver oil preparations had not lost their efficacy through excessive cleaning or coloring processes. The effective substance of cod liver oil is contained in the part of the oil which cannot be saponified. This substance of the oil is a vitamin—vitamin D—which today has been chemically defined. In the classification of diseases, therefore, rickets belongs to the avitaminoses.

Until about 25 years ago two entirely different factors were considered for the development of rickets: lack of light or lack of vitamin D. Exposure to light or a diet including vitamin D containing substances, particularly cod liver oil, cured rickets. Only then was the connecting bridge established between the theories of lack of light and lack of vitamin, through fortunate and bold conclusions. *A. F. Hess* and *Steenbock* and their co-workers were able to demonstrate that substances which in themselves possessed no prophylactic or curative effect against rickets assumed this ability after intensive exposure to ultraviolet rays. Originally ineffective substances exposed to ultraviolet light rays become curative substances against rickets. A great variety of foodstuffs can thus be activated: animal and vegetable oils, milk cream, butter, green salad, starch, various types of flour, etc. Gelatin, paraffin, mineral oil, chlorophyll, pure sugar, zwieback, water and salts cannot be activated. All substances which can be activated contain cholesterol. Ergosterin, which accompanies the cholesterol, is changed into vitamin D_2 when exposed to ultraviolet irradiation. But the cholesterol itself also develops a substance with the same anti-rachitic characteristics—vitamin

D_3. Vitamin D_2 and vitamin D_3 are the real curative substances against rickets.

Thus the circle was completed. The theories of lack of light and of alimentary deficiencies which originally seemed to have no connection could be traced to a common basis. Cure with direct irradiation of ultraviolet light and with medication of vitamin D are, in the last analysis, identical processes.

A cure of rickets can never take place within a few weeks. A disease which produces such major changes of all organs requires several months to heal, provided that those factors which favor the development of rickets are excluded as far as possible. Particularly children who are constitutionally predisposed to rickets show a tendency to recurrence of the disease. Supervision and prophylactic treatment should thus be continued until a time when the growth processes slow down and therefore the tendency to a recurrence of active rachitic symptoms gradually disappears. In the majority of children this limit is reached around the third to fourth year of life. After this age we frequently find still visible remnants of the past skeletal disease which can no longer be removed by the therapeutic measures applied in the cure of active rickets.

The possibilities for the treatment of rickets are as follows:

1) Physical therapy.
2) Nutritional therapy and medication.
3) General treatment with the principal aim of creating a healthy and suitable atmosphere for the child.

ad 1). The best and safest cure of rickets is light which reaches the body of the child as natural sunrays or as efficacious artificial light rays. The effective factor of the light are the ultraviolet rays. the absence of which removes the curative effect of light. This exclusion of ultraviolet rays is accomplished when sunlight traverses glass, mosquito netting or similar substances. Sunbathing behind closed windows is therefore ineffective for curing rickets. Quartz and special types of glass, however, permit penetration by ultraviolet rays with unchanged efficacy. Fumes, fog and dust in the atmosphere also absorb a large part of the effective rays. The amount of ultraviolet rays in the sooty city atmosphere is therefore low, even during the summer months. The sunrays develop their greatest efficacy in the country, in the pure mountain air or on the seashore with its amply reflected light.

> If these variations are taken into account the irradiation with natural sunrays remains the method of choice during the summer months for prophylaxis and treatment of rickets since the effect of light is combined with the influences of fresh air treatment. Light- and sunbathing is applied in form of irradiation of the entire nude body. Irradiation of the face alone does not suffice for curing rickets. Gradation of the irradiation by the sun is necessary in order to avoid damages to the child. In the beginning the infant's entire body is exposed for about two to three minutes in front and two to three minutes on the back, with the head protected from the direct rays, particularly in children who dislike sunbathing. The time of exposure is increased every day by two to three minutes to a total of about 15 to 25 minutes. The speed of increase depends on the skin reaction. Appearance of an erythema is desirable. Severe erythema or inflammation of the skin requires a slowing down, rapid pigmentation permits a longer duration of the sunbath. On warm summer days the infant can remain after the fourth to sixth week of life, lightly dressed or covered, outdoors in the shade for a much longer period of time than in the sunbath, since even the diffuse daylight contains not inconsiderable quantities of ultraviolet light.

In countries with a continental climate natural sunlight is available only on about 100 days during the year in quantities which are sufficient for curing rickets. In subtropical and tropical countries natural irradiation of the sun is adequate for sunbathing on about 300 days or more. In cities with little direct radiation of the sun ultraviolet containing artificial lights replace the natural sunlight. Even with artificial light rays, with so-called mercury vapor lamps (artificial sunrays) healing of rickets takes place with great regularity. With these rays the time of exposure must be even more carefully apportioned than with natural sunrays, with exact observation of the skin reaction of the child. At a distance of 80 to 100 cm. irradiation begins with one minute each on front and back of the body and is gradually increased up to 15 minutes. Application of fat to the skin before irradiation destroys part of the effect of the rays. Irradiation is given three to four times a week depending on the severity of the disease. In general 15 to 20 irradiations with a maximal duration of 15 minutes each suffice for healing rickets. Instead of an excessively prolonged treament more frequent and shorter repetitions are recommended since the efficacy of light treatment depends apparently on the reaction of the skin to irradiation and after prolonged irradiations the skin becomes accustomed so that the effect of the stimulus is reduced. Prevention and definite healing of rickets can be achieved only if irradiation is continued with short interruptions throughout the entire dark season.

Healing as shown in the blood chemistry and in the roentgenogram begins rapidly after light exposure. The changes in the metabolic processes can occasionally cause disturbances in the rachitic child. In some of the children who were suffering from latent tetany in addition to the rickets, the first irradiations occasionally produce a manifestation of the disease in form of laryngospasms or convulsions (see Tetany). If careful examination reveals the existence of latent tetany the anti-rachitic treatment should be combined with anti-tetanic treatment. Rapidly healing rickets in the breast fed infant at times poses demands on the calcium requirements which cannot always be met with a relatively calcium poor diet. It is therefore advisable to give breast fed infants a calcium preparation (e.g., calcium lactate, calcium gluconate, etc.) 3 to 5g per day during the time of the light treatment.

ad 2). There is no diet which in itself is sufficient to cure rickets. On the other hand, unsuitable nutrition which favors the development of rickets should be avoided. A milk intake in excess of ½ liter per day favors the development of rickets. Beginning already with the second or third month of life the daily menu should be supplemented with vitamin C in form of fruit juices, after the third to fourth month vegetables should be given as well as egg yolk which is rich in the anti-rachitic vitamin.

If no light therapy is given the nutritional therapy is supplemented by the medication of cod liver oil which even today is still given occasionally in combination with phosphorus or calcium, for instance:

Phosphorus	0.01	Calc.phosphor.tribas.pur.	10.0
Ol.jecor.aselli ad	100.0	Ol.jecor.aselli ad	100.0

1 teaspoon 2–3 times daily.

Commercially available cod liver oil does not always have an anti-rachitic effect. A pharmacological testing of the vitamin D content of the commercial cod liver oil preparations usually assures now an adequate content of vitamin D_2. A dosage of 10 to 15g cod liver oil (two to three teaspoons) per day suffices for the infant beyond the third month of life.

Among the foodstuffs which can become efficacious curative substances against rickets through ultraviolet irradiation, irradiated fresh milk or powdered milk can be utilized for infant nutrition. The quantity of irradiated milk which is necessary for therapeutic purposes amounts to about 500g daily.

For young infants with a tendency to diarrhea one of the water-soluble vitamin D_2 preparations is recommended. For prophylactic purposes against rickets 400 to 800 I.U. (three to five drops of the customary vitamin D_2 preparations) or two to three teaspoons cod liver oil are sufficient. For the healing of active rickets 8000 to 16,000 I.U. are necessary, depending on the severity of the disease and the age of the patient.

Instead of prolonged daily medication of small quantities of vitamin D_2 one single injection of a larger quantity of a concentrated vitamin D_2 preparation is recommended. Children receive one dose, per os or preferably as intramuscular injection, of 300,000 to 600,000 I.U. of vitamin D_2. This single large dose is particularly recommended under unusual circumstances when a regular administration of cod liver oil or vitamin D_2 cannot be carried out. This single large dose of vitamin must not be repeated until at the earliest two to three months after the first dose (and then preferably not the full dose) in order to avoid the danger of hypervitaminosis D. Damages which occasionally occurred with preparations which formerly were commercially available have now disappeared.

The curative effect of vitamin D_2 takes place rapidly. Already after two to three weeks calcium deposits can be seen in the roentgenogram. The hypophosphatemia also disappears within a short time, and the phosphatase in the serum is reduced.

The same principle which applies to ultraviolet irradiation also applies to vitamin D_2: definite healing of rickets can be accomplished only with continuous medication—with short interruptions —throughout the entire winter. After four weeks of vitamin D_2 therapy a pause should always be made.

ad 3). Apart from light and nutritional therapy a considerable number of curative processes for treatment of rickets have been recommended in the course of the years which have influenced the course of the rachitic disease although not to the same extent as ultraviolet light and vitamin D. This does not include the mostly utopian demand to essentially improve the general living conditions of the child by a hygienically faultless regime. Such a request frequently fails because of economic and social difficulties. The children of the poorest population groups which are most seriously threatened by rickets should from time to time be placed in a children's home. Stays of a few weeks only, however, are of no value if permanent success is to be obtained.

A supporting measure for curing rickets in children after the first half year of life is regular gymnastics and massage which improve the weakness of the muscles and promote the development of static functions. These stimuli act upon the skin and exert a favorable influence upon the metabolic processes in rachitic children. Salt baths which have always been recommended also exert their curative effect in this manner.

There probably is no absolute prophylaxis against rickets. Even with regular irradiation beginning with the first week slight rachitic symptoms such as craniotabes and rosary cannot always be avoided. These symptoms, however, usually disappear if irradiation and vitamin D_2 intake is continued.

The best prophylaxis for rickets is irradiation with ultraviolet rays, by means of sunbaths in summer and artificial light rays in winter. In order to obtain a permanent success irradiation must be continued throughout the entire winter, beginning with the third month of life and in the premature already with the first month of life. It suffices to have the children irradiated two to three times a week. After about 15 sessions there should be an interval of about three to four weeks, after which brief periods of irradiation are resumed. Artificial irradiation will always remain prophylaxis for small numbers. Medication of vitamin D_2 preparations given in small doses over a long period of time or in a single large dose, or cod liver oil are necessary for every infant after the third month of life, particularly during the dark months, in order to prevent the disease.

Hypervitaminosis D

The symptoms which develop from an overdose of vitamin D_2 are characteristic. The children become tired and apathetic. Nausea, vomiting, diarrhea and intestinal spasms develop. The children lose weight. A normocytic hypochromic anemia is observed. Protein, erythrocytes and cylinders are found in the urine. Polyuria and isostenuria develop. The skin becomes withered and dry.

The calcium and phosphorus level of the blood is elevated. Phosphatase is normal or reduced. Non-protein-nitrogen is increased. In the roentgenogram we find enlarged zones of calcification near the epiphyses and occasional periosteal calcium deposits, particularly on the ulna.

After the damage has persisted for a long time anatomopathological findings occur occasionally in form of calcium deposits in the kidneys, in the periarticular tissues, in stomach, pancreas, in the thyroid gland, etc.

Therapy of hypervitaminosis consists of discontinuance of medication of vitamin D_2 in any form, a reduction in the amounts of sun irradiation, ample fluid intake and the avoidance of an alkaline diet. It is assumed that considerable individual predisposition is necessary for the development of hypervitaminosis D_2.

Infantile Tetany

Tetany which even a few years ago was still a common disease of the infant has today become very rare and almost unknown to some of the younger physicians. Tetany of infants, the mechanical and electrical overexcitability of the entire nervous system, was formerly identified with symptoms of rickets so that tetany was at times referred to as "rickets of the nervous system." The differentiation between rickets and tetany was made only much later. No basic similarity between the two diseases can be established because of the differences in metabolism. Even during infancy there are forms of tetany which, just as the tetany in the adult, have hardly any connection with rickets. The absence of tetany in the breast fed infant which is by no means immune to rickets, also supports the theory that apart from the processes which lead to rickets other factors are necessary for the development of tetany. The significance of a disturbance in the metabolism for the development of tetany is also shown in the frequent occurrence of over-excitability in, for instance, the celiac disease. Despite all these exceptions the majority of tetanic diseases during infancy occurs on the basis of rickets. Rickets and tetany are not connected by a blood affinity but rather by a close elective affinity.

The pathological findings of tetany during infancy are a mechanical and electrical over-excitability of the nervous centers and of the peripheral nerves which are occasionally accompanied by obvious pathological symptoms of over-irritability. The stage in which mechanical or electrical over-excitability can be proved only by special testing methods has been described as latent tetany. Manifest tetany is the phase of the disease where objectively perceivable disturbances of the organic functions dominate the picture of the disease.

The existence of mechanical over-irritability is observed upon percussion of any superficially located, peripheral nerve which contains motoric fibers. This can best be demonstrated on the facial nerve where it emerges from the parotis (so-called Chvostek's sign), on the radial nerve on the inner side of the upper arm around the border between lower and middle third, on the ulnar nerve, medially from the olecranon, on the femoral nerve on the anterior surface of the thigh, approximately between upper and middle third. In case of mechanical over-irritability the result of stimulation is a contraction or motion in the muscles which are supplied by the corresponding nerve.

Testing of electrical over-irritability is done through stimulation of a peripheral nerve by a weak galvanic current. For practical purposes the determination of the cathodal opening contraction on the easily accessible median nerve (anode on chest, cathode on the anterior surface of the forearm, near the elbow) is sufficient. A contraction produced with a current of less than 5 milliampere can be regarded as definite indication for the existence of latent tetany.

> In place of the determination of irritability through galvanic current the measuring of the chronaxy has been recommended whereby, in addition to establishing the intensity of the constant galvanic current (rheobase) necessary to produce a minimal contraction the shortest time of exposure to the stimulus is determined which is required to bring about the first muscle contraction with a current of double the intensity of the rheobase. This avoids certain errors. Chronaxy reveals a certain lack of regularity in case of latent tetany which is not observed in the testing of normal nerves. The authors believe to be able by this method to arrive at values which correspond better to the degree of actually present functional disturbances than the determination of galvanic irritability. In practice the determination of the chronaxy has hardly been applied.

Latent tetany could until a few years ago be found in about 60% of apparently healthy, artificially fed infants in form of electrical over-irritability. Today we find less than 10% of children with latent tetany in similar social groups. This decrease in latent tetany together with all the manifestations of the disease is a result of rickets prophylaxis and of better nutritional results with artificial feeding. In spite of this, examination for latent over-irritability, which is easiest done by testing the mechanical over-irritability of peripheral nerves, must be included in the obligatory methods of examination in every infant beyond the third month of life. The borderline between latent and manifest tetany can usually be drawn very closely. The transition from latent to manifest tetany occurs as a result of some new external damage. This can be an infection, a nutritional disturbance, etc. The transition from latent to manifest tetany is connected with considerable changes in metabolism.

The leading symptoms of manifest tetany are tonic-clonic convulsions, laryngospasms and the rare prolonged spasms of single muscle groups, primarily the carpopedal spasms. All these symptoms can develop gradually in every infant in the course of manifest tetany. It is, however, possible to present infantile tetany in three well defined phases each of which is characterized by one of the above mentioned leading symptoms: 1) Before the sixth month of life generalized convulsions dominate the picture of the disease. Twins, prematures and young infants convalescing from severe nutritional disturbances most frequently show this early eclamptic stage of tetany. 2) Around the second half of the first year of life tetany is most frequently observed in form of laryngospasms. They occur invariably in rachitic infants who for a number of different reasons develop the laryngospastic form of tetany. 3) The tonic form of tetany, with chronic muscle spasms dominating the picture of the disease, occurs almost only in children beyond six months of age. These patients usually suffer from severe chronic nutritional disturbances. This form of tetany occurs in atrophic children, in children with chronic dysentery or with chronic digestive insufficiency (celiac disease).

Early eclamptic tetany is never observed in eutrophic full term infants, even if the stage of eutrophy is reached with artificial nutrition. The full term infant develops this form of tetany only when the development of the organism is seriously damaged, either by an acute diarrhea, a chronic nutritional disturbance or an infection causing the changes in the structure of the tissues. The prematurely born infant has a much greater tendency during the third or fourth month of life to this form of tetany beginning with convulsions. The eclamptic form can develop in these children without serious deterioration of the nutritional state only if receiving artificial nutrition. Both groups of children can be protected from the development of over-irritability in the earliest period of life by exclusive breast feeding. Mixed feeding, even if only about one third of the necessary food quantities are replaced by cow's milk, can already cause eclamptic infantile tetany. The particular difficulty in recognizing the convulsions as eclamptic form of tetany exists when the tonic-clonic twitchings persist for days as only symptom, without possibility to prove the existence of mechanical or electrical over-irritability. At times there is even an interval of about eight to 14 days between the period of convulsions and a later period of latent over-irritability, during which time the signs of over-irritability are still absent. This period in which convulsions are present as the only symptom can give rise to a number of diagnostic errors (brain hemorrhage, meningitis). In many cases a diagnosis will be possible only by exclusion, on the basis of characteristic findings of the blood chemistry or on the basis of the definite curative effect attained after placing the infant on exclusive breast milk feeding. In the older infant convulsions due to tetany do not occur without other latent or manifest symptoms of over-irritability. Convulsions in later months of life and in later years, with no other signs of over-irritability, are usually so-called "occasional convulsions" which occur in a number of children at the onset of febrile infections or which may for instance accompany the state of alimentary toxicosis. All these forms of convulsions are not symptoms of tetany. The "occasional convulsions" (febrile convulsions) are the expression of cerebral over-irritability which frequently occurs as hereditary phenomenon or where other symptoms of nervous irritability can be observed in members of the family, or in a small number of cases they might be the first expression of epilepsy. The electro-encephalogram can today contribute to a clarification of the situation.

Laryngospastic tetany was formerly the most frequent form of the disease. With the presence of spasms of the vocal cords mechanical and electrical over-irritability can also be proved in all cases. A few children develop, apart from spasms of the musculature of the larynx, also general convulsions so that we occasionally observe in infants during the fifth and sixth month of life a combination of eclamptic and laryngospastic forms of tetany. Spasms of the glottis lead to temporary apnea but never to asphyxia. The child suffering from laryngospasms is not endangered by closure of the vocal cords but by the changes in the heart which in these children is generally enlarged and has a tendency to sudden arrest due to anatomical and functional damages. In many cases the onset of laryngospasms apparently occurs spontaneously. In other instances the cause can be found in an infection, an excitement, or irritation due to a larger meal. The laryngospastic form of tetany is in close relation to rickets. It never occurs unless active rickets exists simultaneously. On the other hand changes in the state of the tissues, as brought about by nutritional disturbances, do not seem to further the occurrence of this form of tetany.

Apart from the striated musculature of the larynx over-irritability can be shown also on the smooth musculature. Over-irritability of the smooth musculature of the intestinal tract or the urinary tract leads to constipation or to urine retention. The pupils can be of uneven size. Spasms in the musculature of some bronchi lead, through closure of the bronchial lumen, to atelectasis and to secondary infections in the closed off sections of the lungs. A bronchotetany develops which resembles most severe asthmatic conditions.

The tonic form of tetany occurs towards the end of infancy, more frequently during early childhood. Active rickets is not always a prerequisite for its development. The tonic form of tetany with its prolonged spasms resembles in many respects the tetanic conditions occurring in the adult. Carpopedal spasms and tetanic face, caused by spasms of the facial musculature, are the most frequent manifest symptoms of the disease. The smooth musculature is much more frequently involved in this form of the disease than in laryngospastic tetany. General convulsions are not infrequent but laryngospasms occur hardly at all. Signs of mechanical and electrical over-irritability are present from the beginning. Tonic forms of tetany develop when renewed weight drops occur in older children which are chronically poorly developed or when a particularly rapid weight gain sets in during the convalescent period. In all cases tonic tetany is invariably a sign for the existence of more far-reaching alterations in the tissues.

Infantile tetany thus manifests itself in three forms, the symptoms of which can become intermingled. In spite of this it seems justified to maintain this division because of the different origins from which the three forms of the disease develop: in the eclamptic early form—prematurity and disturbances due to acute diarrhea; in the laryngospastic tetany—rickets; and in the tonic form of tetany—chronic nutritional disturbances. In addition there is a definite age limitation with each form of tetany being characteristic for a certain period of infancy. The common denominator of the different forms of tetany is in every case a change in metabolism leading to mechanical and electrical over-irritability which is characteristic for all forms of tetany.

In recent years cases of tetany occurring also in newborns have been described. Shortly after birth, up to the thirtieth day of life, children with normal birth weight who are fed with cow's milk show disturbances in form of vomiting, convulsions, general edema, scleredema, cyanosis and hemorrhages. The disease is apparently more common in male infants. A latent mechanical over-irritability is present in these children but no electrical over-irritability and no manifest signs of tetany. A hypocalcemia (6–9 mg%) is present and in about one third of the patients also an elevation of the blood phosphorus to more than 8 mg%. The phosphatase in the blood, however, remains normal. Tetany of the newborn is caused by a lowering of the calcium level with elevated phosphorus content and normal phosphatase values. The disease develops as a result of frequent vomiting which leads to hypochloremic alkalosis. An insufficiency of the parathyroid glands, perhaps in conjunction with a dysfunction of the as yet immature kidneys favor the development of the disease. The prognosis is good. The symptoms of tetany disappear rapidly with the administration of calcium (e.g., calcium gluconate 2g daily in form of a 2% solution per os) or with intramuscular or intravenous injection of 2 cc of a 10% calcium chloride solution. Prematures which here too are particularly frequently affected receive correspondingly smaller quantities of calcium chloride. As prophylaxis of this rather rare disease it has been recommended to give artificial feedings in form of ⅓ milk with 10% carbohydrates and calcium gluconate. This should avoid the danger of a hyperphosphatemia and a hypocalcemia caused by cow's milk (whole milk, ⅔ milk) which is rich in phosphate with a relatively low calcium content.

A disturbance of the calcium metabolism is the final cause of any infantile tetany. In the blood we find a hypocalcemia as regular manifestation of disturbances in the calcium metabolism. The calcium content of the serum which in healthy infants amounts to an average of 10.2 mg% is reduced to an average of 6.5 to 7 mg% in latent tetany and to 6.0 mg% or less in manifest tetany. The degree of calcium deficit does not correspond to the severity of the disease since irritability depends only on the quantities of ionized calcium. The decrease in calcium ions deprives the organism of the substance which restrains the processes of irritation. Ionized calcium is in its effects similar to ionized magnesium. The antagonists of calcium and magnesium are the ions of sodium and particularly of potassium which increase the irritability. A normal state of irritability can be attained only with a certain balance in the relationship of the alkaline earths (calcium and magnesium) to the alkalis (potassium and sodium). Over-irritability develops when either the quantity of the calcium ions decreases in the blood and the tissues or when the quantity of potassium and sodium ions increases without simultaneous increase of the calcium ions. In most tetanic diseases a decrease of calcium ions seems to take place. Changes in the salt composition in the internal milieu also manifest themselves by the tendency to edema of the tetanic child where we frequently observe taut, edema-like swellings, particularly over the tonically contracted hands and feet.

The pathogenesis of tetany becomes comprehensible if it is known under which conditions the diminished ionization of calcium develops. The task of the therapy is to increase the quantities of ionized calcium in the organism.

The quantity of ionized calcium increases with the acidity of the reaction of the juices as measured by the true acidity. Increase of the bicarbonate and phosphate content of the serum, with a reduction in the concentration of H-ions, brings about a decrease in Ca-ions, or, in other words, an alkalosis of the tissue juices produces a decrease of the biologically active calcium content. A shift of the acid-base metabolism toward the alkaline side is a prerequisite for the development of tetany.

Experimental research has revealed a number of possibilities whereby a displacement of the metabolism toward the alkaline side takes place. Increased breathing leads to so-called breathing tetany because the carbon dioxide content in the blood is strongly reduced. So-called gastric tetany, which occurs in the adult after violent vomiting due to obstruction of the pylorus, develops because large quantities of acid are lost with the vomitus and thus the bicarbonate content of the blood is increased. The same condition and thus the appearance of manifest symptoms of tetany can develop after intravenous injection of larger quantities of bicarbonate. Similar results are obtained with secondary alkaline phosphate.

For our knowledge of the pathogenesis of infantile tetany the experiences with those forms of tetany are of importance where a complete removal of the parathyroid glands was carried out, at first accidentally and later imitated in experiments. The experience that symptoms of tetany develop after complete removal of the parathyroid glands led to the assumption that the destruction of the parathyroids was also the cause of infantile tetany. The occasional finding of extensive hemorrhages in the parathyroids in infants who had died of tetany seemed to support this assumption. But the findings failed to occur with regularity.

The theory of an anatomical damage was replaced by the theory of a functional reduction of the parathyroid glands in case of tetany. The task of the parathyroids is the detoxication or neutralization of metabolic products which develop in the course of normal protein metabolism. Accumulation of the substances in the organism due to absence of the parathyroids leads to symptoms of tetany. Methylguanidine was mainly regarded as the substance which, if accumulated in the organism, causes the development of tetany. Although the picture of guanidine poisoning resembles to some extent that of tetany, there are such considerable differences in the intermediary metabolism that the theory of tetany developing as a result of guanidine poisoning was dropped again.

None of the experimentally produced symptoms which are similar or related to infantile tetany duplicate completely the condition of infantile tetany with regard to appearance, clinical and biochemical findings. Tetany can at first be regarded only as a disturbance of the metabolism, with a hypocalcemia existing as a result of a shift in the acid-base metabolism toward the alkaline side. This produces a reduction of calcium ions in the blood. The consequences of the reduction in calcium ions are aggravated by the simultaneous increase of the potassium and perhaps also the sodium ions. Phosphate, which in case of tetany is relatively or absolutely increased in quantity, also has an aggravating effect upon the irritability.

A stronger irritability of the breathing centers which causes an over-ventilation and thus a reduction of acid valences in the intermediary metabolism promotes the development of tetanic symptoms. This also explains the occurrence of manifest tetanic attacks following violent crying, after awakening from sleep, with temperature rises, etc.

Which influences are known to lead to an acceleration of the metabolism in the infant? The frequent occurrence of manifest tetany in the first weeks of spring was explained by climatic factors which assumedly bring about an acceleration of all metabolic processes. The effect of more intensive sunrays produces so to speak an awakening from hibernation. The term "hormonal spring crisis" has been used. Most of these children show signs of active rickets. Healing of rickets under the influence of stronger sunshine accelerates the metabolism. This fact is confirmed by the observation that the rachitic child, exposed to the effects of ultraviolet rays, occasionally develops symptoms of over-irritability or that symptoms which were already present are aggravated. With the advance of the season the acceleration of the metabolism with the accompanying alkalosis during spring is again balanced. This results in a normal calcium ionization which is necessary for a normal state of irritability.

The fact that only artificially fed infants and never breast fed infants suffer from tetany remains as yet unexplained although breast fed infants are not spared by rickets and are just as much exposed to climatic influences which produce tetany and are not always free from hypofunction of the parathyroids.

> In order to explain these contradictions the larger losses of hydrochloric acid have been considered which take place in the stomach of infants fed with protein rich cow's milk. The more intensive processes of putrefaction in artificially fed children are also said to promote the development of hypocalcemia. The larger quantities of phosphate which are contained in cow's milk are believed to react in the same direction. But no satisfactory explanation has as yet been found.

Similar difficulties are encountered in the attempt to explain the connection of the metabolic processes in rickets and tetany which show such considerable differences, in many respects even opposite processes of metabolism. For the two diseases react similarly toward therapeutic measures.

> The relationship of rickets and tetany is today explained by the theory that despite apparent contradictions in the metabolic processes they are in the final analysis only two phases of a single process, similar to the pendulum which swings across the middle line to the right and left before it comes to rest in the center.

For suppressing the manifest symptoms of the disease a large number of therapeutic processes can be applied which bring about a rapid disappearance of the dangerous symptoms. Suppression of manifest symptoms does not, however, represent the cure of the disease. On the other hand there are a number of curative processes with more gradual effect which cure the disease through normalization of the pathological metabolism. Healing of tetany takes place by way of healing the rickets which accompanies the tetany. Irradiation with ultraviolet rays, the medication of efficacious cod liver oil or of vitamin D_2 preparations are also the treatment for tetany.

The quickest and safest results are obtained by applying artificial ultraviolet light. Already after five to seven irradiations, given in doses which are increased from day to day, the symptoms of tetany disappear. The rise of the calcium level in the blood indicates the onset of healing. The application of ultraviolet light in the therapy of latent and manifest tetany is, however, not without danger. Before any signs of improvement there are not infrequently further increases in irritability of the nervous system. In a child with a hitherto only latent tetany we observe manifest symptoms of the disease after the first irradiations. In place of lesser manifest symptoms laryngospasms or general convulsions may occur. In order to avoid these unpleasant incidences every child with latent or manifest tetany who is to undergo ultraviolet treatment must at first be given one of the symptomatically active preparations which at once suppress the appearance of manifest symptoms of the disease. A calcium preparation, intake of acids etc. (see later) have such an effect. The possibility of the manifestation of a hitherto latent tetany which perhaps had escaped detection, under the influence of ultraviolet treatment makes it advisable to search in every infant who is to receive ultraviolet irradiations for the treatment of rickets, for symptoms of a latent tetany before initiating the irradiations.

With other therapeutic measures such as cod liver oil, vitamin D_2 etc., the healing process is less rapid. There the adjustment in metabolism takes place so gradually that there is no perceivable aggravation of tetany. By regular intake of 10 to 15g daily of an efficacious cod liver oil or vitamin D_2 in the customary dosages tetany can be cured in the course of two to three weeks. Exclusive feeding with breast milk—although breast milk contains only small quantities of vitamin D—also heals manifest tetany with certainty and even severe convulsions cease, particularly in young and prematurely born children. In those cases cow's milk must be excluded completely from the diet. After three to four weeks of exclusive breast milk feeding all symptoms of tetany have disappeared. The physician will recommend this method of treatment particularly for infants who show signs of tetany already around the time of the third month of life.

An adequate diet serves to support symptomatic treatment. Large meals should be avoided since any excessive filling of the stomach aggravates the symptoms of over-irritability. The quantity of cow's milk in the diet should be reduced. If severe manifest symptoms appear older eutrophic infants receive only tea and sugar for 12 to 24 hours. Caution is indicated in the use of such a hunger therapy in older atrophic children who usually develop symptoms of tetany with continuous convulsions. Following the period during which only tea is given small quantities of food (400 to 500g per day) are offered. In older infants all food substances can be given, such as vegetables, meat, fruit, with the exception of cow's milk whey which is the tetanogenous substance. It is recommended that liquids be given in form of cereal water to which about 1% flour, 2% butter, 5 to 7% sugar and 1 to 2% of one of the commercially available calcium-protein preparations or amino-acid preparations have been added. The child can take about 400 to 600g per day of this mixture. Cow's milk must also be avoided or used only in smallest possible quantities in the preparation of cereal. Cooked farina or rice, mashed potatoes prepared with water must be substituted for several days until the curative effect of anti-tetanic preparations becomes apparent. Tetanic symptoms occurring in an early stage of convalescence from an acute diarrhea, in an atrophic child or in a hydrolabile infant make such an extensive exclusion of milk inadvisable. Milk mixtures which are poor in whey such as protein milk should be recommended in such cases. A particularly intensive application of other symptomatic anti-tetanic preparations is then necessary in order to compensate for the increase in irritability emanating from the small quantities of whey.

Because of the alkalosis which is present in cases of infantile tetany acidified milk mixtures have been recommended such as hydrochloric acid milk, consisting of 600 cc whole milk mixed with 400cc n/10 hydrochloric acid or 960cc whole milk with 40cc n/1 hydrochloric acid. After addition of 5 to 7% sugar the child can be given the quantities and mixtures which correspond to his age and nutritional state. Hydrochloric acid milk has only a symptomatic effect, which suppresses the alkalosis but does not cure the basic disturbances of metabolism in tetany. Ready-to-use preparations of this type have become commercially available.

Other symptomatic measures which suppress the alkalosis are the administration of ammonium chloride which is given as 10% solution (0.5 to 1.0g per kg) dissolved in larger quantities of milk. Also, the action of calcium is today interpreted as the action of an acid. Anorganic calcium salts are more efficacious for these purposes than the organic compounds. The most frequently applied preparation is calcium chloratum siccum which is given in amounts of 5 to 6g daily in cases of manifest tetany. From a 10% solution the child receives one large teaspoon full with every meal. In order to make the unpleasant taste of this preparation more palatable it can be prescribed in the following form:

Sol. calc. chlorat. cryst.	30.0/250.0
Liq. ammon. anis.	3.0
Gummi arab.	2.0
Syrup simpl. ad	300.0

1 large teaspoon 5 to 6 times a day

More effective than the chloride of the calcium is the calcium bromate which combines an acidifying with a sedative effect (calc. bromat. 10.0, Aq. dest. ad 100.0, 1 large teaspoon three to four times daily). A balance of the disturbed equilibrium between alkalis and alkaline earths can be accomplished with magnesium and strontium salts which are recommended for the treatment of manifest tetany. Magnesium sulfate (of a 25% solution 0.2 gm magnesium sulfate per kilogram of body weight intramuscularly) is applied in severe states of over-irritability of the smooth musculature (bronchotetany) which endanger the life of the patient. All these medications—hydrochloric acid, calcium, magnesium—show their effect only after several hours. In all severe and dangerous manifestations of tetany such as clonic-tonic convulsions, severe laryngospasms, rapidly reacting sedatives and narcotics must be given immediately in order to remove the danger of life. Urethan (1g), chloralhydrate (0.5g), Luminal (0.05 to 0.1g) and similar medications given, if necessary, in repeated doses, suppress the serious manifestations of tetany. Urethan and chloralhydrate are best given in form of small retention enemas (dissolved in 25cc cereal water), luminal in form of sodium luminal as intramuscular injection. The medication of these preparations is to be continued until the effect of the slowly reacting symptomatic preparations sets in. This requires six to 12 hours. This second group of symptomatic medications must in turn be given until the influence of the real curative measures of tetany (ultraviolet irradiation, vitamin D_2, human breast milk) become effective. This requires several days in cases of manifest tetany.

In latent tetany the exclusive application of purely symptomatic medication is not indicated. This only suppresses a warning symptom. The finding of a positive facialis phenomenon or of an electrical over-irritability in the infant makes it necessary to apply one of the approved curative measures for tetany.

This holds true to a certain extent for the prophylaxis of tetany. The timely application of an anti-rachitic curative measure in the artificially fed child or feeding on the breast are the safest means of prophylaxis against tetany.

For not quite apparent reasons latent and manifest tetany has today become a rare disease. The widely spread prophylaxis against rickets does not suffice to explain the decrease in the occurrence of tetany. Even children who suffer from active rickets develop only very occasionally symptoms of latent or manifest tetany. The severe disturbances of the entire nutritional state, particularly in the older child (celiac disease) are also much more rarely accompanied by tetany than in previous years. Education concerning a suitable diet, primarily the avoidance of feeding excessive quantities of milk, the realization of the value of light, air and sun for the child have perhaps contributed to the disappearance of tetany.

ANEMIAS OF INFANCY

The fact that nutrition is of decisive significance for the development or the curing of some of the anemias of infancy justifies a short description of this condition in a book dealing with the theory and practice of infant nutrition. Placing this chapter among the nutritional disturbances has also its justification because in most of these anemias deficiencies in nutrition and absence of substances known to be of etiological significance in anemia can be proved. Here too establishment of an adequate diet is an essential part of therapy. It is of significance that severe nutritional disturbances during infancy such as the severe form of gastro-enteritis or severe damages of the nutritional state (atrophy) only rarely involve the hematopoietic organs to any larger extent. This depends in part on the age predisposition of the different forms of anemia. The severe forms of anemia which belong here occur more frequently after the first year of life while primary nutritional disturbances are dominant in the first half year of life. Anemias are observed more frequently only at the time of appearance of the severe cases of rickets, scurvy, tetany, repeated infections and of secondary dystrophies which accompany all these conditions.

The three forms of anemia which occur during infancy and are directly or indirectly connected with nutrition are:

a) Anemia of prematures and twins.
b) Nutritional anemia.
c) Anemia due to infection.

The severity of the different anemias can vary in all forms. Severe and light stages of the disease, diseases with or without splenomegaly are observed in the etiologically different groups. Blood picture and bone marrow findings also do not permit any judgment regarding the etiology of an anemia. Different causes can lead to similar pathological changes in the peripheral blood and the bone marrow. The following stages of changes in the blood picture should be differentiated:

α) Hypochromic anemia, characterized by a strong decrease of the hemoglobin, with almost normal or normal number of erythrocytes. This results in a color index which is lower than 1. Except for minor poikilocytosis and basophilia there are no pronounced changes in the red blood picture. The white blood cells also appear normal with regard to number and composition. The bone marrow is unchanged.

β) Normochromic anemia without splenomegaly, with minor symptoms of irritation in the blood. In these patients hemoglobin and number of blood corpuscles are reduced to approximately the same measure, resulting in a color index of about 1. Findings of the red blood picture point toward a state of irritation or of exhaustion in the blood forming organs. Basophilic erythrocytes and a few young forms of erythrocytes are observed. The white blood cells do not reveal any definite changes in number or composition as long as there are no infections.

γ) Normochromic or hypochromic anemia with splenomegaly and changes in the blood picture pointing toward severe functional disturbances in bone marrow and liver. The number of erythrocytes is reduced to 2 to 3 million, more rarely to even lower values. The hemoglobin content is lowered correspondingly so that the color index of 1 persists. Not rarely, however, the strongly anisocytotic blood contains, similar to pernicious anemia, large erythrocytes which are rich in hemoglobin (megalocytes) resulting in a color index above 1. In these conditions and not only in the rarely occurring aplastic form of the disease a number of pathological cell forms are found in the red blood picture: polychromasia, basophilic stippling, normoblasts and megaloblasts can be observed. The white blood cells also appear involved. Even in cases without infection there is a moderate leukocytosis, the mononuclear cells are increased in relationship to the neutrophil cells. Myelocytes, even in larger number, are also observed occasionally in the peripheral blood. In the severest cases of anemia the number of platelets decreases, the protein content of the blood serum is reduced, and the hematocrit value is lowered. In addition, some patients develop changes in the bone marrow with an increase of megaloblasts with moderate hypoplasia, more rarely hyperplasia of the marrow.

The degree to which an anemia progresses or the form in which it appears depends in part on the particular etiology, but more upon the degree of damage and primarily upon the constitution of the child. All three stages can sooner or later appear in all of these anemias. It is important to emphasize this point since there is still a tendency to consider the severest stages of anemia which are accompanied by marked splenomegaly and considerable blood changes and which somewhat resemble the changes occurring in pernicious anemia, as separate diseases and to differentiate them from other anemias of infancy. So-called anemia pseudoleucaemia infantum, *Jacksch-Hayem's* anemia, is merely the most advanced stage of an infantile anemia which can originate from any cause.

Anemia of Prematurity

A more or less severe degree of anemia develops in every prematurely born child and in a large number of twins even if they are full term babies. These children are never anemic at birth. At the time of birth hemoglobin and number of erythrocytes are normal. In the course of four to eight weeks there occurs a rapid drop in the hemoglobin content to 10 to 12g (40 to 60%) and of the number of erythrocytes to 4.0 to 3.0 million. The resulting anemia is frequently hypochromic. At times the prematurely born infants do not appear as pale as would correspond to the hemoglobin content and the number of erythrocytes. In the majority of prematures the physiological anemia can, with careful nursing care and nutrition, be overcome by the fourth to sixth month of life even without special therapy.

There is no agreement regarding the cause for these anemias. A storage deficiency of iron might be possible since the infant cannot meet his iron requirements during the period of exclusive milk feeding with the minimal quantities of iron contained in the milk. During the first months of life he is therefore dependent on the iron depots which were stored only during the last months of pregnancy. In later months of life infants receive the necessary quantities of iron in a mixed diet (vegetables, fruit etc.). A histological examination of spleen and liver of anemic prematures shows, however, that these organs contain an abundance of iron which originates from destroyed erythrocytes. There is no real "internal" iron deficiency. In contrast to full term infants, the function of blood formation in the marrow of prematurely born infants is still immature and undeveloped, resulting in a disproportion between deficient new blood formation and physiological destruction of blood. This insufficiency can, however, not be noticed in the bone marrow. The marrow is rich in cells and rather hyperplastic. It is particularly rich in cells of the red series (normoblasts, erythroblasts). As in some of the other forms of anemia the delivery of erythrocytes from the marrow is disturbed also in these cases. An improvement of the anemia is shown in the peripheral blood as an increase in the number of reticulocytes. If it is assumed that the iron deficiency is the essential cause of anemia in prematures, this form of infantile anemia would belong among iron deficiency anemias (see later). The assumption that an immaturity in the functions of the hematopoietic organs is of greater significance places this anemia in a special position.

Of more practical importance than a temporary physiological anemia of prematures are the later anemias of these children which develop from the so-called physiological anemia of prematurity. They even develop occasionally in cases which suffered no external damages such as frequent infections or faulty nutrition. More frequently an adjustment of a physiological anemia of prematurity is prevented by infections or nutritional disturbances. As in any form of anemia, the predisposition of the patient seems to be of significance also for the development of anemia of prematurity. The predisposition determines the degree to which the anemia develops or whether the later months of life bring a transition to a hypochromic anemia. This second phase of anemia of the premature takes place around the fifth to sixth month of life. The feeding difficulties and lack of appetite which are frequently observed in these children promote the development of this form of anemia. The children then assume a yellowish pallor. They are not dystrophic, frequently even fat. The severity of anemia varies from the lightest degrees to the fully developed symptoms of anemia resembling pernicious anemia. Finally, if the anemia is not cured, all these children develop dysergia and the inability to fight off infections presents the greatest danger to these children who at this period of life show signs of immaturity also in other functions.

NUTRITIONAL AND INFECTIOUS ANEMIAS

Pure Nutritional Anemia

The purely nutritional forms of anemia of the infant, which undoubtedly occur, are of less practical significance because they develop less frequently than those forms of anemia which are caused by nutrition and infection. Pure nutritional anemias during infancy have become rare. They develop at the end of the first year of life, more frequently during the second year, in infants who were fed with goat's milk, in infants who were fed exclusively with breast milk for too long a time and in infants who received large quantities of cow's milk without addition of other foodstuffs. The absence or deficiency of vitamin C and iron which is contained in vegetables and fruit is one of the causes of nutritional anemia which occurs as a result of any one-sided milk diet. In addition there is a deficiency of folic acid resulting from an insufficient intake and deficient ability for utilizing the folic acid. As a result of damage and inadequate development of metabolic processes which are necessary for the formation of this substance, folic acid is not placed at the disposal of the organism in sufficient quantities. However, here too a constitutional tendency toward anemia must exist. The number of infants who thrive even on a faulty diet and who do not become anemic is not inconsiderable.

Any pure nutritional anemia leads to dystrophy and to dysergia. The dystrophy is at times little pronounced or concealed through deposits of spongy, pasty fat. Dysergia is never absent in the long run from the picture of nutritional anemia of infancy.

Bronchitis and pneumonia develop in these pale poorly eating children.

Nutritional and Infectious Anemia

Faults in nutrition and frequent infections are revealed in the histories of almost all anemic infants. However, it cannot always be determined in the individual case whether the development of anemia was caused more by an infection or more by nutritional disturbances. Similarly it is not always clear whether faulty nutrition prepared the ground for the ensuing infection or whether an infection caused damages in the nutritional state and nutritional processes. In the further course of the disease the two damages appear in any case intertwined to such an extent that it becomes hardly possible to determine whether the anemia was caused and continued through infections or rather through deficiencies in nutrition. The state of the anemia is aggravated by any new infection. Any accumulation of infections is in turn accompanied by diarrhea, vomiting, lack of appetite and thus by a further deterioration of the nutritional state. A sharp differentiation between nutritional and infectious damages cannot be carried out in these cases.

The anemic conditions which occasionally accompany rickets are also nutritional and infectious anemias. If anemia is frequently observed in severe cases of rickets we find, apart from deficiencies of light and vitamin D_2 also other nutritional and infectious damages. The assumption of a special anemia of rachitic children is unjustified.

The nutritional as well as the nutritional and infectious anemias are usually of the hypochromic type. The patients are pale and show a lack of appetite. Functional heart murmurs can be heard in severest stages of anemia even during infancy. In the blood we find anisocytosis, microcytosis and frequently a leukopenia with relative lymphocytosis.

INFECTIOUS ANEMIA

Purely infectious anemias occur during infancy primarily with lues and tuberculosis. The severest degrees of these anemias are reached when for instance a child suffering from tuberculosis develops in addition a number of grippal infections. In lues the disease itself causes anemic conditions. The tendencies of ordinary bacterial infections and of virus infections to cause anemia can be proved at any time in the infant if hemoglobin values and erythrocyte counts are determined at the onset and at the end of a period of elevated temperatures, even if it lasts for only a few days. The decrease in red blood cells and hemoglobin brought about under the influence of grippe, chickenpox and similar conditions is, however, compensated again within a short time provided that the infection which had just been overcome is not followed by a new disturbance such as a nutritional damage. Macrocytic anemias are particularly frequent in prolonged infections.

The effect of bacterial-toxic damages on the blood picture can be observed during infancy in almost every disease which is accompanied by fever. But only few infections produce a prolonged or chronic anemic reaction. Apart from lues and tuberculosis which were already mentioned above, stronger and more stubborn anemic conditions result particularly from pyurias and septic staphylococcic and streptococcic diseases.

Etiology and pathogenesis. The problem as to how anemia develops under the influence of various causes (prematurity, nutritional damages, etc.) can be answered about as follows:

1) Through a primary deficiency of iron or other specific components of the hemoglobin, e.g., deficiency of pyrrhol compounds or of folic acid.

2) Through a weakness or immaturity of the blood building functions of the bone marrow or in the infant also of the liver.

3) Through direct nutritional damage.

a) Through toxic substances in the food which lead to a dissolution of red blood cells resulting in a pathologically elevated blood destruction (trophotoxic anemia).

b) Through deficiency of certain food substances which are necessary for normal functions of blood formation (trophopenic anemia).

4) Through damage of blood formation by bacteria and bacterial toxins, whereby the damage is accompanied by a deterioration in the utilization of iron in the intestines.

In the development of anemia the disturbances in the formation of hemoglobin and the disturbance in the formation of blood cells do not run parallel. An inadequate hemoglobin formation is accompanied by a disturbance of bone marrow functions as well as a disturbance in the activities of the liver. A disturbance in the formation of the blood cell structure is essentially a sign of damaged activity of the bone marrow. The dissociation of the two tasks is proved by the presence of blood changes with approximately normal or normal number of erythrocytes and with relatively strong reduction of hemoglobin quantities (hypochromic anemia) and the occurrence of anemias with a stronger reduction in number of erythrocytes than corresponds to the hemoglobin content (hyperchromic anemia).

In the pathogenesis of anemia primary iron deficiency is of main significance. A deficiency in iron intake occurs when the child is fed over a long period of time with milk which contains very little iron. During the first months of life this lack of supply is compensated by iron reserves which are present in full term infants. The age predisposition of anemia and the particular tendency of prematures to anemia, since they could not build up iron reserves due to premature birth, seemed to find their explanation in this manner. The lack of efficacy of prophylactic or therapeutic intake of iron in organic or anorganic form in prematures speaks against the theory of iron deficiency. Similar to rickets which is never cured by calcium intake alone, a large number of anemias cannot be cured by iron intake since the organism is unable to utilize this iron supply for hemoglobin formation. Only after the ability for new blood formation is re-established and substances other than iron which are necessary for hemoglobin formation (pyrrhol compounds, folic acid, vitamin C) are available the ingested iron can be utilized for hemoglobin formation.

The iron reserve which is present at time of birth lasts in the full term baby only to the fifth or sixth month of life. A one-sided milk diet beyond this time (see chapter on damages due to excessive milk intake) or delay in the addition of iron to the food (e.g., damages due to excessive flour intake) leads to anemia. Exclusive feeding with breast milk beyond the sixth to seventh month of life also fails to give the child the iron quantities necessary for hemoglobin formation which amount to about 0.5 to

0.7mg per kilogram of body weight per day (1 liter breast milk contains about 2mg iron). Exclusive feeding with cow's milk also produces iron deficiency. This is aggravated by a lack of vitamin C which, with a simultaneous iron deficiency, promotes the development of anemia; these two deficiencies occur almost always simultaneously with an insufficient intake of iron in form of fruits and vegetables. Anemias resulting from a vitamin C deficiency are not rare in the second half of the first year of life. This is not always accompanied by scorbutic symptoms. Anemia develops already with an inadequate vitamin C intake which however is not sufficiently pronounced to bring about signs of scurvy.

The theory of a weakness or immaturity of blood formation applies to all forms of anemia. It is the manifestation of the constitutional basis which must be present for the development of anemia. It explains why under similar external conditions only a small number of children become anemic. The majority of prematurely born infants show an immaturity of the blood forming functions so that they usually all become anemic, at least temporarily. The maturation of blood forming functions takes place in most infants and in a number of premature children before the age of six months. The immaturity is not only a functional unfitness of the bone marrow.

The diet of the infant can bring about a direct damage of the blood. The observation that the development of anemias during infancy is promoted by exclusive milk feeding led to a search of the damaging substance in the milk, that is, in the milk fat. Fatty acids which in artificial nutrition are absorbed in large quantity were believed to cause an increased blood decomposition which could be proved by changes in the bilirubin metabolism. There are as yet no final proofs for such a trophotoxic effect of food, particularly of milk. Trophotoxic and trophopenic damages probably play a role in the development of goat's milk anemia. The low vitamin C content of goat's milk and the abundance in goat's milk fat of fatty acids which at least in vitro have a hemolytic action explain this peculiar position of goat's milk anemia with regard to its pathogenesis. However, the experience that despite continued feeding with goat's milk anemia can be cured or prevented if the diet is supplemented in other directions speaks against the assumption of a primarily active damaging effect of goat's milk.

The prognosis for a cure of anemia was not favorable in former years. The then available curative measures brought about only a very slow improvement of the pathological changes in the blood. They exerted no influence upon the numerous kinds of infections which sooner or later developed in the majority of anemic children. Progress was accomplished only when it became possible not only to remove rapidly the pathological blood findings but simultaneously to obtain a favorable influence on the general condition and on the dysergia. Treatment of anemia during infancy and early childhood has since become an easier task. Therapeutic progress was attained in three steps: the first step was the proof of the significance of an adequate nutrition for the curing of anemias. The second step brought the systematic application of blood transfusions. The third step was taken when American authors recommended giving liver and liver extract as well as folic acid for the cure of certain forms of anemia. Which of these three therapeutic measures are to be applied in the individual case depends on the form and the severity of the anemia and upon the degree of involvement of the general condition of the patient. Nutritional therapy is necessary in every anemic patient. In children beyond the first year of life liver therapy can be included by adding liver to the child's diet. Treatment of anemia with blood transfusions should be applied in those cases where it is important to avoid acute danger by rapid correction of the blood picture. Transfusions are thus indicated above all in patients who had been suffering from anemia for a longer period of time and in whom an accumulation of serious infections had caused danger to their life. Blood transfusions are the method of choice also in all acute anemias which are accompanied by a far-reaching destruction of blood (e.g., favism). Transfusions should also be attempted in cases where other therapeutic measures had brought no significant improvement of the blood picture after two to three weeks.

The following table gives indications for the treatment of the different forms of anemia (after *Carl H. Smith*):

Size of Cells	*Quantity of Hemoglobin*	*Therapeutic Agent*
Normocytic	normal	transfusion
	reduced	iron
Macrocytic	normal	liver & folic acid
	reduced	liver & folic acid & iron
Microcytic	normal	transfusion
	reduced	iron

Nutritional therapy of anemias of infancy beyond the fourth month of life and of early childhood begins with greatest possible reduction of milk intake. Milk is replaced by a mixed diet which also includes meat, liver and purées from other inner organs such as spleen and lungs. A mixed rich diet exceeding the quantities given to healthy children is the basic dietetic treatment of anemias in the second half of the first year of life. Any one-sided diet with a preponderance of milk and milk mixtures should be avoided. The following diet can be recommended for a child of about nine to 12 months:

First meal: Malt coffee or tea or cocoa made with water, with sugar and a little milk; dark bread with butter, jam or honey.
Second meal: fresh fruit (bananas, apples) and fruit juices.
Third meal: a plate of fresh vegetables with mashed potatoes or boiled farina, with two to three tablespoons of puréed meat, preferably liver; plus about 50 to 100g fresh fruit juice;
Fourth meal: Malt coffee, tea or cocoa with sugar and a little milk, and cookies.
Fifth meal: Dark bread or black bread with butter or margarine, soaked in about 100g sweetened whole milk, with four to eight teaspoons of white cheese or cream cheese in any form; fresh fruit. One egg.

It is possible to improve or cure light and medium cases of anemia within six to eight weeks with a dietetic treatment without any drug therapy. Children suffering from dysergia and from more severe forms of anemia must also be given the more rapidly acting curative medications apart from nutritional therapy. A liver therapy, consisting of 50 to 100g boiled or steamed liver, results in a surprisingly rapid cure of anemia. Liver therapy brings about at first a rapid increase in the number of erythrocytes, while the increase of hemoglobin values follows somewhat slower. Liver offered in form of a purée is frequently rejected by anemic children with a lack of appetite. It is somewhat easier, provided the culinary abilities of the mother are sufficiently advanced, to supply these amounts of liver hidden in puddings and desserts. In view of these difficulties it is advisable to recommend efficacious liver preparations which are available in liquid or powder form or in form of injections. 15 to 20g daily of the liquid extracts suffice for the infant. In the course of three to four weeks the majority of children show a normal blood picture without necessitating the application of other therapeutic measures.

Blood transfusions can be reserved for those anemic conditions where healing is delayed, where the anemia is severe and for patients who suffer frequently from intercurrent infections. Today a blood transfusion has become a relatively simple procedure in the hospital. In children the transfusion is performed intravenously. Infusions into the sinus longitudinalis which were formerly performed at times, or the occasionally still recommended intraperitoneal infusions are not advisable. The infusion can be performed upon freeing of a surface vein (elbow, ankle region) or, even in the young infant, through puncture of a vein in the skull or in the back of the hand, the lower arm, etc. The jugular vein can be clearly seen with the head hanging down; however, this vein slides easily so that a puncture is not always successful without ample experience.

The quantities of blood required for transfusion depend on the degree of anemia. If the blood which is utilized for the transfusion is normal with regard to hemoglobin content and number of erythrocytes, 15cc of the donor's blood are necessary per kilogram of body weight of the recipient in order to raise the number of erythrocytes of the recipient by 1 million per cubic millimeter. If for instance an anemic infant with a weight of 7 kilogram and an erythrocyte number of 2 million per mm^3 is to have the number of blood cells increased to 3 million, it requires a transfusion of $7 \times 1 \times 15cc = 105$ cc of the donor's blood. In order to avoid coagulation of the donor's blood, 10% of a $3\frac{1}{2}\%$ sodium citrate solution is added to the blood used for the transfusion. In the above mentioned example requiring the transfusion of 105cc of blood, 10cc of the sodium citrate solution would be necessary; this is placed into the sterile container which serves as the receptacle for the donor's blood. The utilization of fresh blood seems more advisable than the utilization of older blood from a blood bank, particularly since usually larger quantities of some preserving fluid are added in order to preserve the blood. If such blood is utilized part of the preserving fluid should be poured off before the transfusion.

In order to avoid accidents a determination of the blood type of donor and recipient is required. A transfusion is permitted only when the blood serum of the recipient does not agglutinate the blood cells of the donor. For this purpose drops of blood serum from persons whose blood type is known or of one of the commercially available test sera are placed on a glass slide. A complete determination includes the application of serum of group A, serum of group B and, to be certain, also serum of group O. A drop of blood the type of which is to be determined is added to each one of these three drops. The result corresponds to one of the pictures presented in the following table. According to the following pattern the blood type is determined depending on the presence or absence of blood agglutination. If agglutination takes place in all three drops (serum A, B and O) the recipient has a blood type AB. If serum B and O are agglutinated the recipient belongs to blood group A, etc. If test serum A and test serum B are used alone four different reactions take place which also permit a classification of the recipient. The utilization of a third serum O increases the certainty of differentiation.

Blood Type of Person to be Examined	*Test Serum* *A*	 *B*	 *O*
AB	x	x	x
A	o	x	x
B	x	o	x
O	o	o	o

x = agglutination, o = no agglutination

If the blood groups of donor and recipient have thus been established it can be determined on hand of the second diagram whether a transfusion is permitted or not. "+" in the table signifies agglutination, meaning a transfusion is forbidden; "–" signifies the absence of agglutination and thus permission to carry out the transfusion.

Donor	*Recipient (Serum)*			
(Erythrocytes)	*AB*	*A*	*B*	*O*
AB	—	+	+	+
A	—	—	+	+
B	—	+	—	+
O	—	—	—	—

In order to reduce still further the danger of damages caused by transfusions so-called cross-agglutination must be carried out in every case. A small quantity of blood is taken from the recipient and centrifuged. The serum from this centrifuged blood is tested with the erythrocytes of the donor for the presence or absence of agglutination. A transfusion is permitted only when there is no agglutination in these tests. Because of the danger of sensitization of Rh negative infants through the transfusion of Rh positive blood in case of repeated transfusions as well as for later pregnancies the determination of the Rh factor is of significance. For Rh negative infants only Rh negative blood may be used for transfusion.

The result of the transfusion is an immediate increase in hemoglobin and erythrocytes, corresponding in number almost exactly to the quantity of transfused blood cells. This rapid improvement of the blood picture is proof that the transfused erythrocytes remain in existence in the recipient for some time. However, since these erythrocytes, like all other blood cells, have only a limited life expectancy but, on the other hand, anemia is definitely cured after a few transfusions, it must be assumed that the transfusion exerts a stimulus which re-establishes the damaged hematopoiesis. The individual transfusions are given in intervals of three to eight days, depending on the severity of the anemia and the results of repeated blood tests, until approximately normal values are reached for hemoglobin and erythrocytes. Apart from the improvement of the blood findings the results of the transfusions become evident in a better appearance of the children which is already noticeable during the transfusion, in an improvement of appetite and mood, in the influence upon the state of dysergia and at times in an immediate weight gain.

For intramuscular injections which must always be regarded as emergency measures only, citrated blood is used in quantities of 20 to 40cc which are deposited in one or two places. A determination of the blood group is not necessary in such cases. The transfused blood cells are destroyed after the transfusion. Only their component parts are absorbed and act as basic materials or stimuli for new blood formation.

Drug therapy of anemia during childhood has lost significance due to the more rapidly acting therapeutic measures such as liver and intravenous blood transfusions. Perhaps this is unjustified. An appropriate iron therapy, particularly in conjunction with an intensive nutritional therapy, can produce favorable results in hypochromic anemias. In those cases the effect of the iron seems to take place only after the change in nutrition has eliminated the damages to the blood formation. Iron therapy futhermore seems to be useful in all those anemias which were cured in some other manner, in order to maintain and further develop the results obtained. Iron is effective only if given in large quantities because of the poor absorption of iron from the stomach and upper jejunum, even in the healthy infant. The iron preparations chosen should if possible be ferrous compounds which are absorbed better than ferric iron preparations. In calculating the necessary iron quantities the relatively large iron requirements of the infant must be considered (0.5 to 0.7 mg per kilogram of body weight).

All iron preparations easily and frequently cause in the infant vomiting, lack of appetite and diarrhea. These disturbances frequently make an adequate iron therapy in form of ferr. reductum, ferr. sulfuricum, etc. impossible. However, a great number of commercial iron preparations are available, for instance in combination with ascorbic acid or copper or cobalt, which are better tolerated than the plain iron salts. The use of preparations which also contain copper or cobalt does not seem absolutely necessary for the treatment of iron deficiency anemias.

Folic acid is indicated in macrocytic anemia in childhood which developed as a result of faults and deficiencies in the diet, possibly in combination with vitamin B_{12}, 10 to 30γ, three to six injections. During infancy this applies practically only to those anemias which occur in the course of celiac disease where the absorption of the anti-anemic factor is disturbed. Amounts of 5 to 10mg of folic acid per day, given per os, are required. Similar to these conditions are macrocytic anemias which develop in infants during the second half of the first year of life who are suffering from gastro-intestinal disturbances and from diseases of the air passages. The bone marrow reveals an abundance of megaloblasts while leukopenia and thrombocytopenia exist in the peripheral blood.

The prognosis for anemia in childhood is today relatively favorable. Even severe anemias can be cured. The time necessary for curing anemias can, by utilizing all available therapeutic measures, be estimated at about four to six weeks.

Prophylaxis of anemia has also been improved through recent findings. A complete mixed diet including liver and liver preparations offers protection against the development of anemias. The observation of these nutritional requirements is particularly important in prematures in order to prevent the transition from physiological anemia to the clinically more serious late anemia. Prophylaxis of anemia with iron therapy fails in premature and full term children unless the absorption of iron is improved with a diet which is rich in vegetables and unless the processes of new blood formation are normalized. A complete diet, which in time is supplemented by as rich and as varying a diet as possible, is also necessary for avoiding repeated infections.

CHAPTER VIII

Diarrheal Diseases

DIARRHEA, its origin, its clinical significance and its cure has always been the main problem in the theory and practice of infant nutrition. It has been attempted to pursue in theory the different ways, which even today are not fully clarified, in which a diarrhea may develop. The practical task is to rapidly and safely cure the diarrhea. Diarrhea itself endangers the child through loss of water, salts and nutritional substances. A correct diagnosis and adequate therapy of diarrhea protect the child from the dangers of dystrophy and atrophy.

Many problems have not as yet been solved in theory. In practice every case of diarrhea, with very few exceptions, can today be cured. Treatment of diarrhea in infants is never quite simple. There is no pattern for therapy which can be applied to every patient suffering from diarrhea. Treatment of diarrhea has always required and still requires today, despite all progress, a considerable amount of individual attention. The impossibility of establishing a pattern for the treatment of acute, subacute and chronic diarrheas makes instruction in this field very difficult. Only extensive practical experience will give the physician the security to offer suitable advice in the individual case.

The difficulties in evaluation and treatment of diarrhea are threefold:

1) The causes for diarrhea in the infant are numerous and at times not clear even for the experienced practitioner. Completely opposing damages are among the origins of diarrhea. Diarrhea develops from excessive food intake. But the fact that also a deficiency in food, either of the total quantities or of individual food substances, can lead to diarrhea is less known. While in the first case the diarrhea is caused by surpassing the tolerance, a dys-

trophic condition which develops particularly rapidly in hungry newborns and infants leads in the latter case to a lability of intestinal functions and thus to diarrhea. In other cases an infection in the intestinal tract or an infection occuring in the organism, far removed from the digestive system, can become the cause for a diarrhea. It is by no means rare that the cause of the diarrhea cannot be explained at all, especially if the diarrhea has existed for a longer period of time and has already resulted in a deterioration of the nutritional state.

2) The effects of diarrhea on the entire organism of the child are numerous and varying. Cases of diarrhea of similar severity and similar character can in one child be accompanied by an undisturbed general condition and good development. In another child a similarly or even less severe diarrhea can bring about a most serious condition, abrupt weight losses and definite disturbances in the general condition.

3) Treatment required for this same symptom, diarrhea, also varies considerably. In some cases a temporary interruption in food intake and a reduction in food quantities become a therapeutic necessity; in other cases an unchanged continuation of the previous diet is to be recommended; in still other patients an increase or supplementation of the diet must be prescribed despite an existing diarrhea.

Such a survey is bound to cause confusion to a logically thinking person. Any basis and system of therapy to be chosen is apparently lacking. But the above contrasts were not listed for the purpose of causing confusion. Only a physician who attempts to recognize the numerous causes leading to the symptom of "diarrhea" can finally understand the different diagnostic evaluations and the varying treatment of diarrhea. In order to treat diarrhea adequately a careful analysis of the entire symptom complex is imperative in every case. We must again and again call attention to the fact that diarrhea itself is only a symptom which frequently, however, fails to give indications as to the diagnosis and the necessary therapy.

The clinical definition of the symptom of "diarrhea" is known. The term diarrhea is applied when number and composition of stools deviate from the norm, when the stools become more frequent and more liquid, that is, contain more water. With this change the pasty consistency of the normal stools is lost. Stools are less compact, frequently torn into small pieces and shreds. At other times a watery, greenish-yellow liquid is eliminated which contains only a few more solid particles. Large amounts of mucus appear and the normal color of the stool is changed. Frequently it is of a greenish color already upon elimination or it assumes this color in the diaper within a short time. The alkaline reaction of the stool, as found usually in the artificially fed infant, is changed as shown in tests with dampened litmus paper. The reaction of the stool becomes acid. Occasionally, however, an alkaline reaction may appear, especially in most severe diarrheas. This comes about when abundant quantities of alkaline reacting mucus and intestinal juices are eliminated with the stools with strong irritation of the intestinal mucosa and rapid peristalsis so that the acid end products of digestion are neutralized in the artificially fed infant and finally surpassed by the alkaline substances.

Beyond these easily perceivable changes no essential data for the practical treatment of diarrhea have been observed by more profound analyses of the diarrheal stools. This applies to microscopic as well as to chemical examinations and to the results obtained with the methods of physical chemistry. By none of these methods has it been possible to characterize a diarrhea to an extent which would provide indications for therapy. For some time it was believed that microscopic examination of the stool would reveal the damaging agent the removal of which from the diet would cure the diarrhea. If for instance an abundance of fat drops or fatty acid crystals were found in the stool the conclusion was made that fat was not well tolerated; if a large number of so-called casein pieces, which are white tough particles of the size of peas or even beans, were found in the stool it was claimed that the ability for casein digestion was damaged. All these symptoms have today assumed another meaning. Any increase in peristalsis results in increased quantities of fat in the stools. If it is possible to slow down the peristalsis, with a similar fat intake but with other changes in the diet, the "fat-diarrhea" ceases. This applies similarly to findings of undigested flour in the stool, from which in former years serious conclusions were drawn on the necessary composition of the diet. A positive iodine test, that is, blue or blackish-blue discoloration of the stool upon addition of a few drops of tincture of iodine containing free iodine was said to prove the inability of flour digestion. But here, too, the appearance of starch in the stools is only the result of an acceleration of the intestinal passage. In this manner part of the flour escapes digestion and absorption. Raw milk which already in the stomach begins to produce rough curds, produces curds in the stools which escape decomposition through the protein-splitting gastro-intestinal ferments, particularly with an accelerated peristalsis. With regard to the reaction of the stools it has already been pointed out that alkaline reactions can exist even in severe diarrhea while normal stool eliminations and harmless diarrheas with a cow's milk diet produce not infrequently an acid reaction.

Great effort and labor have been applied to bacteriological analyses of diarrheal stools. Attempts were made again and again to isolate specific pathogenic germs in diarrheas of infants. The streptococcic enteritis of which one used to speak was perhaps the first result of these studies. The tabulation of typical pathogenic coli strains as causes of serious diarrheas presents the latest attempt to determine etiology and clinical evaluation of diarrhea through bacteriological examinations of the stools (see later). However, all these examinations, the significance and necessity of which should in no way be doubted, have not led beyond the findings encountered upon observation of the stools at the bedside.

With the failure of all attempts at a finer characterization of the stool the physician will direct his attention beyond the pathological symptoms in the gastro-intestinal tract to the considerably varying and variable symptoms in the entire organism of the child. The exact examination of the stools does not permit a decision regarding the nosological position of diarrhea and thus an indication for its treatment. Only the condition of the child, the appearance and intensity of certain pathological symptoms and, if possible, a determination of the causes of the diarrhea permit a correct diagnosis and thus an indication for adequate therapy.

This leads to a further question: Are there changes in the appearance and the functions of the child which permit a characterization of the diarrhea to the extent which would make possible a correct evaluation and treatment of the disease?

As was emphasized already above, there are children who show no physical or psychological disturbances with the onset of diarrhea. On the other hand there are others who the day before still appeared active and with a healthy color and who show severe signs of illness with the onset of diarrhea. The changes in their appearance and behaviour are so pronounced that even the mother or the nurse must perceive the change in their general condition. These changes occur not only in the functions of the gastro-intestinal tract. They affect the entire organism and all its functions. Only the presence or absence of such changes permits a classification of the conditions which accompany the frequent eliminations of stools. A classification can be made immediately into: a) Diarrheas with disturbances of the general condition, and b) Diarrheas without disturbances of the general condition.

The elimination of a normal stool is dependent upon a peristalsis the speed of which is well regulated, upon a gastro-intestinal secretion which guarantees the complete decomposition of the ingested food, upon an extensive absorption of the ingested food and finally upon the presence of a bacterial flora which is stabile in locality and in type. In addition there are processes taking place beyond the gastro-intestinal tract. The normal or pathological products of a disturbed digestion which are absorbed from the intestines can exert a damaging effect anywhere in the organism, in the liver. the central nervous system and finally, in any one of the cells. The diversity of functions which contribute to the stool formation explains the many different ways in which a diarrhea can develop. Central nervous influences bring about an accelerated peristalsis. Examples of such a central irritation are those diarrheas which accompany any kind of infection. Another time a change in the gastro-intestinal chemistry, as occurring with overheating and with hunger—in both cases the secretion of intestinal juices is reduced—is the cause for the diarrhea. On the other hand, primary disturbances in digestion and absorption of food during infancy seem to be rare, according to our present day knowledge. And finally, the biology of the intestinal bacteria is of decisive significance in the pathogenesis of diarrhea. For diarrheas which are accompanied by disorders in the general condition the disturbance of the bacterial life in the intestines is today the focal point in the search for pathogenesis and therapy of diarrheal diseases.

Depending on their location, quantities and type, the bacteria in the intestinal tract of the infant are of decisive significance in determining the digestive processes. As long as the bacterial flora in the intestines remains within normal limits and as long as only the customary physiological types of bacteria occur in the permitted areas the digestive activity remains undisturbed. The intake and settling of certain types of bacteria within certain sections of the intestines is one of the most important tasks which the newborn has to fulfill after birth. By what means this task is accomplished successfully in most cases is beyond our knowledge. It is essential that the peristalsis is regular and of normal speed; this prevents accumulation of intestinal contents but on the other hand leaves sufficient time for accomplishing the absorptive processes. Perhaps there are antibody-like or bacteriostatic forces present in the intestines which make it possible to exclude unwanted bacteria from one or the other section of the intestines and to permit settling of bacteria only in areas where they can be of use to the digestive process. In the duodenum such bacteriacidal substances or substances which impede bacterial development (so-called bacteriostanine) have been proved. The healthy organism fulfills the task of regulating bacterial life in the intestinal tract with admirable precision. The bacterial flora which settles in the stools of breast fed infants is therefore uniform but again different from that settling in the stool of children fed with cow's milk. The organism of the host determines not only the type of bacteria which are permitted to enter and settle in the intestines, even the sections of the intestinal tract where the bacteria live and develop are exactly determined already in the newborn. In those parts of the intestines where the important fermentative and physico-chemical processes of digestion and absorption of food take place the bacteria and thus their participation in food decomposition are excluded. The duodenum and the entire small intestines are practically free of bacteria. Bacteria are limited almost exclusively to the large intestines where the chemical fermentative digestion has already been completed. Any disturbance of the balance of this arrangement of the bacteria and any invasion of bacteria into territories of the intestines from which they are normally excluded cause severe disturbances in the organized activities of food decomposition. The disorder in the settling of the intestines is the first and immediate cause of a diarrhea. However, we still lack the knowledge as to the cause for the appearance of new pathogenic types or as to what factors bring about the ascent of the germs customarily found in the large intestines into regions of the small intestines for which they are not destined (see also p 337).

The descent of normal intestinal inhabitants from the stomach into the small intestines or the ascent of "physiological" germs from the colon into the small intestines is less frequently the cause of diarrhea than the invasion of new, more or less pathogenic germs. In the intestines of healthy, artificially fed infants we find in 90% of the children Escherichia coli. In addition we find in 70%, alone or together with other bacteria, enterococci and in 50% aerobacter aerogenes. Apart from the coli strains for which an attempt is made at present at a classification into special groups, there are types of coli with supposedly pathogenic characteristics. These types, which in former years were occasionally described as dyspepsia-coli, are today referred to partly as paracoli or, according to their characteristic serological behaviour, as pathogenic coli types.

These coli strains with special biological characteristics are believed to be of significance in the development of acute diarrheal diseases. Paracoli belongs to the enterobacteria which are gram-negative germs which ferment slowly and which can be characterized by their ability to be agglutinated by certain antisera. There is probably a biological relationship of these species to the salmonella groups and perhaps to the proteus groups. The following bacteria are included today in the paracoli group: bacillus paracoli aerogenes, the paracolon bacterium and the bacillus paracoli anaerogenes, according to their ability to ferment sugar with or without gas formation. These germs, which also occur in a considerable percentage of healthy children (carriers?) can become pathogenic. Paracoli bacilli are also frequently found in water and milk. A separate species, found in eggs, has been described as paracoli arizona.

In individual epidemics of diarrhea and in a few patients with acute gastro-enteritis it was believed that special strains of b. coli could be isolated which were described as D.433 or Escherichia coli D.433 L and as 433 β, and which are characterized by a special antigen structure by which they can be differentiated. For example: Escherichia coli D.433 L has an O (Soma) antigen 111 and a K (capsule) antigen B 4. Mobile forms have in addition an H antigen which seems to occur in the flagellae and is named "2" or "12." Thus for the coli strains D.433 L. frequently found in acute diarrheal diseases an antigen formula 111 B.4.2 or 111 B.4.12 is characteristic. The Escherichia coli 433 β has an antigen structure of 55.B.5.6. These coli strains are not sensitive to sulfa preparations and to penicillin. However, they might be sensitive to streptomycin, chloromycetin and aureomycin. These complicated bacteriological examinations of some of the "pathogenic germs" of acute gastro-enteritis are by no means completed. We have as yet no definite proof that these coli strains with special antigenic characteristics are in fact the causative organisms of acute intestinal diseases. They occur frequently in the stools of patients with acute gastro-enteritis, but are also not rarely found in the intestines of healthy infants, particularly those living in the environment of a sick infant. Escherichia coli 111 B.4. and 55 B.5 are found occasionally as the only germs in the stools. At other times we find in addition: species of paracoli, proteus mirabilis, proteus aerogenes or pseudomonas aerogenes. But above all it has so far not been possible to prove the presence of antibodies in the serum of patients against all these bacteria; only this would furnish the final confirmation for the pathogenic nature of these germs.

Germs of the shigella and salmonella groups are found in increasing measure in cases of diarrheal disturbances. The frequency of the findings varies in the different countries depending on external hygienic conditions and on the extent of the presence of these germs in the surroundings of the child. At an average there are indications of 20 to 25% for the occurrence of bacteria of the shigella group and about 5% of the salmonella group. All of these organisms cannot be found in the blood of the patients. It is occasionally possible to prove antibodies in the blood serum for the organisms of the salmonella group and more rarely for those of the shigella group. For paracoli, proteus and pyoceaneus the serological proof of such substances in the blood has so far succeeded in only a few cases.

Whether virus infections (with the exception of occasional epidemics) are of significance in the common acute diarrheal disturbances remains as yet only a hypothesis. Whenever proof of such virus infection was attempted it was successful only occasionally in acute intestinal diseases (see chapter on diarrhea of the newborn), and the significance of these positive findings for the development of the disease seems in no way certain.

At present the conclusion is accepted that a serious condition develops in the intestines as soon as the settling of bacteria ceases to be limited to the areas of the large intestines or the stomach but extends in greater intensity to the small intestines. In all cases of diarrhea where the small intestines are not involved or in any case the action of the bacteria does not extend over larger areas of the intestinal wall, there are no extensive changes of the general condition of the child. The theories concerning the varying pathogeneses of the symptom of "diarrhea" force the physician to regard the elimination of frequent liquid stools only as a symptom which can appear as part of a great number of diseases. The symptom of diarrhea can be evaluated correctly and purposefully only when the physician concerns himself not with the pathological form of the stool but with the presence or absence of pathological changes in the general condition which he must regard as the leading symptom for the diagnosis and therapy of the diseases in question.

DIARRHEAS WITH DISTURBANCES IN THE GENERAL CONDITION OF THE CHILD

If the elimination of frequent liquid stools is accompanied by signs of a disturbance of the general condition, if diarrhea occurs together with other pathological symptoms, we speak of a diarrheal disease. Acute diarrheal diseases can at least didactically be described in two forms which, however, cannot be sharply differentiated with regard to their etiology and pathogenesis. The terminology of the two pathological conditions has changed in the course of the years. While the diseases used to be commonly described as acute dyspepsia and as alimentary toxicosis or intoxication (*Finkelstein*) it seems more suitable at present to speak of lighter and more severe forms of gastro-enteritis or to differentiate between acute diarrhea and severe and toxic gastro-enteritis.

In acute diarrhea or acute dyspepsia the clinical symptoms of diarrhea and disturbances in the general condition occur to about the same extent, meaning that despite disturbances in the gastro-intestinal tract the disturbances in the general condition are relatively light. In the case of intoxication, of severe and toxic gastro-enteritis, however, the serious disturbances in the general condition dominate the picture almost completely. The diarrhea, on the other hand, although occasionally of particular violence, becomes of less significance. This differentiation is of particular importance for the urgency of therapeutic measures. In acute diarrhea the treatment of the loose stools is of prime consideration.

It therefore seems indicated for practical purposes to separate the relatively light acute gastro-enteritis (synonym: acute dyspepsia and infantile diarrhea) from the severe form of diarrheal disturbances, the toxic or severe gastro-enteritis (synonym: acute intoxication or toxicosis). While in acute diarrhea the development of disturbances does not progress beyond the first stages, in cases of toxic gastro-enteritis they continue to progress from relatively harmless beginnings. The results are severe functional disturbances in all organic systems, particularly in water and salt metabolism, in the circulation, the vegetative nervous system and the central nervous system. An acute diarrhea is in almost every case the beginning of a severe form of disease. By overlooking this first phase of the disease or by giving inadequate treatment acute diarrhea will develop into toxic gastro-enteritis.

The transition from the light form of the disease to the severe form is promoted and accelerated by a number of external circumstances. Factors promoting this transition from acute diarrhea to toxic gastro-enteritis are: development of the disease in infants during the first three months of life, unsatisfactory nutritional state, special (hydrolabile) constitution of the child, specific infections of the intestines or severe parenteral infections. In older infants or in eutrophic children the disease develops only into an acute diarrhea, provided there are no other external damages. It is possible that one physician might diagnose only a light form of diarrheal disturbances while another physician will regard this same case already as a severe gastro-enteritic intoxication. The frequent diagnosis of "beginning intoxication" or "subtoxicosis" shows the difficulties which at times confront the physician. In the interest of therapeutic action the physician should attempt to reach a definite decision. In doubtful cases it is of advantage to the patient if those measures are prescribed and applied which are indicated for the treatment of the more severe form of the disease.

The views regarding the etiology of acute gastro-enteritis have changed in the course of the years. While originally the bacterial infection of the intestines was placed in the foreground in considering the etiological significance, the nutritional development of acute gastro-enteritis was subsequently emphasized. At present the theory that bacterial processes are of decisive significance in the development of the disease is again supported. It is possible that milk which was already strongly contaminated with bacteria was ingested by the infant or that species of bacillus coli with particular biological characteristics can cause an acute gastro-enteritis. Much can be said in favor of the theory of an exogenous infection of the gastro-intestinal tract with bacillus coli, bacterium proteus, pyocyaneus germs which hitherto had been regarded as not or only slightly pathogenic. The possibility of such an infection of the small intestines is particularly great in dystrophic and atrophic children who no longer possess the ability, as eutrophic children do, to control or destroy invading germs of any kind.

Apart from the invasion and settling of new germs which hitherto had been foreign to the child, there is also the possibility of an endogenous infection of the small intestines through the normal intestinal germs. An example for this is the large number of cases of acute gastro-enteritis which occur in the course of or together with acute parenteral infections the organism of which (pneumococcus, staphylococcus, etc.) cannot be found in the intestines. In these cases the normal intestinal inhabitants can be observed in the duodenum and the small intestines which are disturbed in their functions through the primary disease.

The causes of acute gastro-enteritis, listed according to their frequency of occurrence, are: 1) Parenteral infections (particularly in young children, see p. 455). 2) Overfeeding with a diet of correct or incorrect composition leads to stasis in the intestinal tract (that is, to disturbances in the peristalsis). If the child receives excessive amounts of an incorrectly balanced diet there will, in addition, develop an aphysiological course of the intestinal processes, similar to that occurring in any type of faulty nutrition (over-feeding with sugar, flour or fat). 3) Infected or decomposed food, with pathogenic bacteria, e.g., salmonella or special species of the coli groups or other germs occurring in the milk. Mass infections with such germs can no longer be checked by the intestines. All these germs can, in addition, cause secondary changes in the milk which affect the functions of the small intestines. The different types of pathological germs and even normal intestinal inhabitants then find in the damaged small intestines a terrain for multiplication and expansion. The acidification of the milk itself is, in such cases, of little significance. In any case it has not been possible so far to prove that milk which curdled spontaneously through the action of the customary intestinal bacteria can become the cause of acute diarrheal disturbances. This also corresponds to the fact that strongly acidified milk mixtures are valuable curative means in the treatment of diarrhea (e.g., buttermilk, protein milk, lactic acid milk).

For practical purposes of artificial nutrition an improperly acidified milk is, however, not suitable for infant feeding since the changes in the milk could have been brought about also through pathogenic bacteria or through a mass infection of the milk and since occasionally peptonizing bacteria can cause the development of toxic products of decomposition of protein (peptones). Infected or decomposed food is only of little significance as cause of a gastro-enteritis in institutions with a careful management of the milk kitchen and also in the private home where only clean and correctly handled milk or milk powder is used. Under unhygienic, unfavorable external conditions many acute diarrheal diseases develop in this manner during the hot summer months. At times it might be difficult to determine whether the acute gastro-enteritis developed only as a result of feeding the infant with spoiled food or whether other external damages such as excessive heat, unfavorable environment etc. did not contribute to the development of the disease through their harmful effects. High temperatures act on the child in a similar manner as on the food.

4) Overheating of the child can bring about acute diarrheal disturbances. It has, however, not yet been definitely established in which ways the heat can bring about the development of

diarrhea and damages to the general condition of the child. Factors which must be considered are: a reduction of the secretory functions of the gastro-intestinal tract or disturbances in the production of substances, as yet little known, which safeguard the absence of germs in the small intestines, nervous influences upon the peristalsis, or an influence of the temperature, which becomes elevated due to overheating, upon the intestinal activity.

5) A rapid cooling off can in sensitive children bring about an acute diarrhea.

6) Any inadequate care of the infant can become the cause of an acute gastro-enteritis if accompanied by one of the above mentioned damages.

In surveying the causes which lead to an acute gastro-enteritis primary causes which can by themselves produce a gastro-enteritis can be differentiated from secondary causes which merely promote the development of acute diarrheal disturbances. Primary causes are: parenteral and enteral infections, including not only specific infections such as dysentery, paratyphoid etc.; over-feeding, and the intake of infected and decomposed food. Secondary causes are overheating, faulty care of the child and rapid cooling off.

It appears indicated to consider separately the pathogenesis of diarrhea and the pathogenesis of general disturbances which develop in the course of an acute gastro-enteritis.

Under the influence of one of the causative factors disturbances develop in the normal peristalsis. This results in a stasis of the intestinal content which in turn prevents the physiological, regular and complete emptying of the small intestines during the intervals between meals. This provides quasi a bridge of food remnants for the bacteria over which the germs can invade the small intestines from the stomach or from the large intestines. Changes in the fermentative processes which under the influence of severe infections or excessive overheating occur particularly readily in children with a disturbed nutritional state, similarly bring about a displacement of the intestinal germs. It might also be that specific substances, so-called bacteriostanines, which have the ability to control the bacterial growth in the duodenum, are lost.

Once one of the above mentioned factors has brought about a settling of bacteria in the small intestines, far reaching changes occur in the intestinal chemistry. While for instance carbohydrates are almost completely digested by the intestinal ferments and rapidly absorbed, they serve as medium for the bacteria in infants whose small intestines are invaded with germs. The digestion of carbohydrates through bacteria takes place now in a different manner and leads to different end products than the physiological fermentative decomposition of sugar and flour. The substances which now develop from the carbohydrates cause stimulation of the peristalsis. In the ensuing acceleration of intestinal passage more and more carbohydrates reach the lower sections of the intestines which are most densely settled with bacteria. Furthermore the carbohydrates are so to speak the favorite food for those types of bacteria which most frequently occur in the intestines. If fats are also contained in the food they, too, promote bacterial growth. Even the protein can finally not escape the attack of the bacteria and by undergoing a faulty decomposition it contributes to the increase in the small intestines of nonphysiological products of decomposition of the food. Simultaneously with the ascent or descent of bacteria into the small intestines their peristalsis is accelerated. The thickening of the fecal matter now fails to take place. There is no time for normal water resorption from small and large intestines. The irritation of the intestinal wall is shown by the presence of mucus in the stools and in the microscopically discernible appearance of leukocytes in the feces.

The symptoms outside the gastro-intestinal tract which occur with great regularity in every acute gastro-enteritis, are:

a) Loss of body weight.
b) Disturbances in temperature regulation.
c) Disturbances in the general condition of the child.

The speed and amount of weight losses of several hundred grams and more within a few days cannot be explained with the loss in organic substance alone. A child with acute gastro-enteritis cannot lose protein or carbohydrates or even fat from the tissues or from the small reserve depots of nutritive substances in such a short time and in such large quantities. The weight loss in acute diarrheal diseases represents essentially a loss of water.

The results of examinations of the entire metabolism in acute gastro-enteritis confirm this assumption. Children with less severe forms of diarrheal disturbances show neither losses of salts nor negative nitrogen balances. Acute dyspepsia resulting from infection presents a certain exception since in those cases disturbances occur in the metabolism of mineral substances and of protein which are a direct result of the infection and the fever. Water losses have also been proved through continuous weighing of the child. The water losses, however, are not due to the increased elimination of water in the liquid, loose and frequent stools. They are caused essentially by the increased water elimination through lungs and skin. In severe cases considerable quantities of salts are also lost (see later).

The water which is lost in acute diarrhea originates essentially from the extracellular water depots, in severe cases also from intracellular fluids. Certain factors, which as yet are not completely known, disturb the normal ability of the tissues to bind water in a certain form, in certain places and in certain quantities.

> This substance could assumedly be hyaluronic acid or one of the related substances. The hyaluronic acid is a protein free mucopolysaccharide which occurs in the tissues in a very viscid solution and is considered the basic substance of the tissues and therefore also called "tissue cement."

These and other substances of an unknown nature assure in the healthy child the fixation of water in the tissues and in the cells. The water loss from tissues and cells ceases as soon as the pathological intestinal processes are removed through adequate treatment. The safest way to accomplish this is the exclusion of all food and the administration of only water per os or parenterally (see later). With the elimination of the damaging substances causing the water loss which originate in the diseased intestines, the weight decrease comes to a standstill, despite the hunger, and not infrequently we even observe the resumption of water retention on this water diet. The weight decrease in children with acute gastro-enteritis is not the result of hunger. It is, on the contrary, basically connected with the intake of food into the intestines which for some reason have been damaged. But even the symptom of diarrhea is not the decisive factor for the onset of water loss. For while diarrhea accompanied by weight loss exists in the acute stage of dyspepsia, weight gain frequently sets in already after introduction of a therapeutic diet at a time when the loose stools still persist in undiminished severity and with unchanged appearance of the feces. In infants whose water binding in the tissues is a priori less solid due to constitutional anomalies (so-called hydrolabile children, see p. 514) any acute gastro-enteritis represents a serious disease since in these cases the loss of water from the tissues and perhaps also from the cells takes place more rapidly and in larger quantities than in infants who are constitutionally normal with regard to their water binding capacity. This applies also to children during the first three months of life. At this age infants suffer from a physiological hydrolability which in the majority of children disappears only in the course of the first three months of life. Thus any acute diarrheal disturbance at this age becomes a serious disease. Similar dangers due to non-stabile water binding exist in any acute diarrheal diseases of dystrophic and particularly of atrophic children. Many of these children have a hydrolabile constitution. External damaging factors (hunger, frequent infections, faulty nutrition, etc.) have contributed to more or less destroy the ability for water retention. The aim of any therapy in acute diarrheal disturbances must therefore be a stoppage of the water losses. This approach to the treatment is more important in the first phase of acute diarrheal disturbances than the attempt to cure the diarrhea. For practical application the measures taken for curing diarrhea and for stopping the water losses are frequently identical.

The amount of water losses in acute diarrhea remains in most cases within moderate limits, as can be concluded from the weight decreases. Any larger water losses which then include also large quantities of vital water deposits, particularly also intracellular fluid, are characteristic for toxic gastro-enteritis or intoxication.

The causes which lead to a mobilization of intercellular and intracellular fluid are not known to us. Perhaps the intestinal wall is damaged by the pathological processes in the gastro-intestinal tract so that it permits permeation of aphysiological "toxic" substances. The substances which were considered in this regard were mainly the toxic products of decomposition of protein (amines, polypeptides).

No definite proof could as yet be established concerning the existence of an abnormal intestinal permeability, either through histological examinations of the intestinal wall or through the finding of amines outside the intestinal tract in blood or urine.

The absence of evidence of increased intestinal permeability in acute diarrheal diseases led to the assumption of damages in the liver functions through disturbances in the digestive processes. This should bring about disturbances in the catabolism and anabolism of protein and formation of toxic products of decomposition outside the intestines in the liver. But here too there is no indication as to which of the numerous functions of liver activity contribute to the process of water loss. The findings of a cloudy swelling and of fatty degeneration of the liver as well as the results of the liver function tests are so far the only basis supporting the theory of the significance of liver damage in the pathogenesis of acute diarrheal disturbances. This again brings up the question whether these are not secondary changes occurring as a result of primary disturbances at a different place. The vegetative nervous system has been considered as such a place which, damaged by toxic substances, was regarded as point of origin for all the disturbances which as a total represent the picture of acute diarrheal diseases and which, in its most severe forms, leads to the symptoms of a neuro-vegetative shock.

There is finally also the possibility that as a result of primary disturbances of food digestion in the intestines, substances which are of significance for the processes of normal water absorption and retention in the cells are no longer absorbed in sufficient quantities. In this respect one might consider primarily an abnormal digestion of carbohydrates which as we know are of importance for physiological water absorption. There are no basic proofs for any one of the theories concerning the pathogenesis of water losses in acute diarrheal disturbances. Observations which have been made so far merely permit the conclusion that a disturbance in the functions of water retention exists.

In the first stage of the disturbance the effect manifests itself in a state of thirst, in a dehydration of the organism (exsiccosis simplex—*Fanconi-Rossi*). Insufficient fluid intake, overheating, concentrated food, vomiting and diarrhea cause a disturbance in the fluid balance. The losses are no longer balanced by corresponding intake. The water which is drawn upon by the patient in order to equalize the disturbed balance originates essentially from the extracellular fluid deposits which represent about one fourth to one third of the entire fluid in the body of the infant (about 700 to 1500 cc). With the water also salts, sodium, chlorine and bicarbonate are lost.

The losses in water do not essentially take place through the loose stools but, as mentioned above, mainly through the increased insensible perspiration through skin and lungs. So far it is essentially a pathological aggravation of physiological processes which occurs with the onset of a diarrheal disturbance. If diarrhea is accompanied by vomiting, losses of chlorine ions and of acid valences take place which result in a metabolic alkalosis. In the blood the organism attempts to replace the chlorine losses through mobilization of Na_2CO_3.

As a result of losses in water, salts and alkali or acid ions the acute gastro-enteritis brings about disturbances in the acid base metabolism depending on the extent to which disturbances in breathing, diarrhea and vomiting contribute to the pathological picture. Dehydration caused by an inner thirst (cell or tissue thirst) leads to an increase of electrolytes in the plasma (hyperelectrolytemia) or later, depending on the displacements in the water depots, to hypoelectrolytemia.

> If in hyperelectrolytemia the values of Na and Cl are calculated for normal values of plasma protein (that is, water content of the blood) the resulting values for sodium and chlorine are, even in hyperelectrolytemia, below normal. This determination is of practical significance. It shows that even in a relative hyperelectrolytemia the therapeutic intake of water and salt is indicated (*Freudenberg*).

If the water losses continue they may lead to a thickening of the plasma in which the fluid content can no longer be maintained. The water reserves in the three large fluid depots exist no longer in physiological quantities. Signs of blood thickening in this second stage of the developing "metabolism catastrophy" which is characteristic for acute toxic gastro-enteritis, are: increase in number of erythrocytes, of hemoglobin and rise in hematocrit value. The sodium chloride content of the plasma is now elevated (chloride: 340–380 mg%). This thickening of circulating fluid leads to disturbances in circulation and to a contraction of the arteries of the skin. The patients appear very pale. The results are an insufficient blood supply of kidneys and brain. On the other hand there are no signs as yet of disturbances in the acid-base balance: the alkali reserves are still normal. The blood thickening brings about changes in viscosity and flow of the blood. The processes of cell breathing suffer. The oxygen intake in the cells is reduced. The slowing down of cell functions contributes to an accumulation of acid metabolic products, resulting in a metabolic acidosis. The alkali reserve drops to values below 40. The normal reaction of the blood of pH 7.5 is lost. The pH of the urine drops to below 5.5. As a result of circulatory disturbances in the kidneys a condition can develop which resembles the so-called lower nephrone nephrosis. There is oliguria, occasionally increased to a state of anuria and an elevation of urea in the blood. The condition which has now been reached can best be described as a state of shock. Continuation of the damaging influences leads to increased permeability of cell and capillary walls. The result is a syndrome which resembles a "serious inflammation." A hypoproteinemia develops. The salt content of the serum is now elevated due to the failure of the excretory functions of the kidneys. Ingested water flows off through the capillaries and is lost in large quantities through pathological, deepened and accelerated toxic breathing.

We can thus summarize the following stages in the development of a toxic condition:

First stage: Only water losses (e.g., through heat damages). Protein and water content in serum normal. As a result of the water losses the salt content in the body fluids increases.

Second stage: Extracellular water and salt losses. (Total protein, erythrocytes, hemoglobin are elevated.) The blood is thickened.

Third stage: Extra- and intracellular water and protein losses. Condition resembles a serious inflammation. An anhydremic, anoxemic condition exists.

Graphically the complicated processes are presented in the following diagram (from *Fanconi-Rossi, Freudenberg*).

Further details will be found in the chapter on the pathogenesis of toxic gastro-enteritis (see p. 390).

Control of urine secretion, of number of erythrocytes, of hemoglobin and of the hematocrit are the simplest methods of determining objectively the extent of blood thickening and of circulatory disturbances. The determination of the alkali reserve and of the chlorine content in the serum is a valuable supplement for gaining a picture of the situation of the metabolism and thus indications for necessary therapy. These few data furnish approximate information regarding the situation of the entire metabolism.

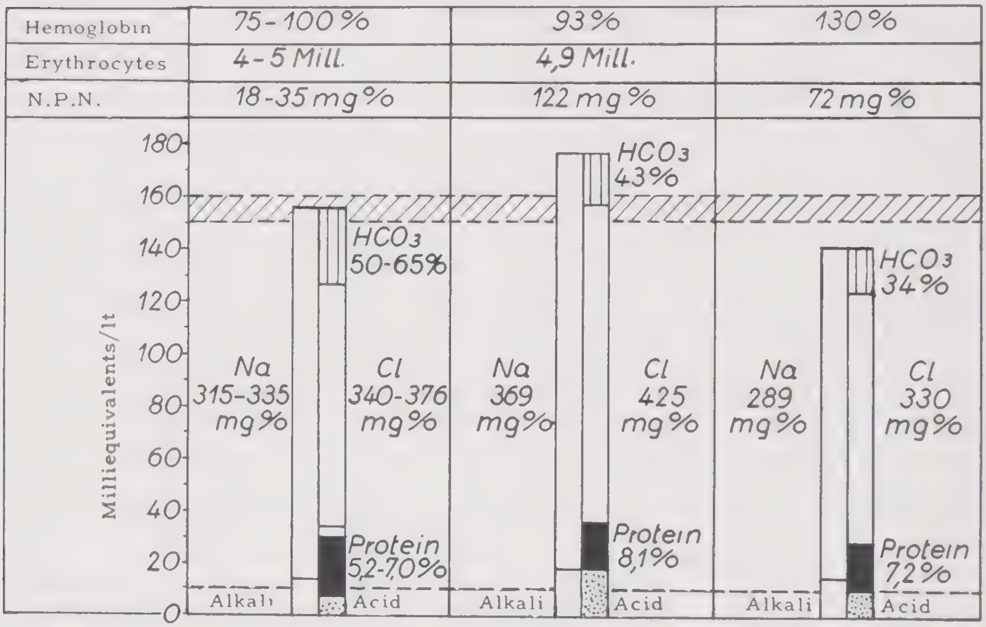

Fig. 32. Ionogram (*Freudenberg* and *Franconi-Rossi*). a) Normal ionogram; b) Ionogram in dehydration (hyperelectrolytemia), thickening of the blood, moderate degree of acidosis; c) Ionogram in dehydration and severe diarrhea. More severe thickening of the blood, reduction of alkali reserve and metabolic alkalosis.

Also the potassium participates in the migration of water and salts from the cells into the extracellular tissues and into the plasma and, vice versa, from the extracellular space back into the cells. The significance of this displacement and the losses of potassium have been particularly stressed in the last years (*Darrow et al.*).

Potassium is found in the organism, as mentioned already above, mainly in the cells. In the extracellular fluids potassium is contained only in relatively small quantities. The potassium contained in the intestines is almost completely re-absorbed by the healthy gut. Normally 90% of the potassium is eliminated in the urine, and only 10% in the stools. In severe acute diarrheal diseases an increased quantity of potassium is released by the cells resulting in a hypopotassemia in the cells as well as in the serum. The organism attempts to compensate for the low potassium level in the cells by absorption of sodium chloride from the blood into the cells.

The pathological symptoms connected with the loss of potassium depend on the potassium losses in the plasma. Pathological symptoms resulting from hypopotassemia appear when the potassium content of the serum drops below 8–10 mg% (= 2.05–2.56 milliequivalent).[*] Those pathological symptoms which appear as a result of acidosis and hypopotassemia will be described later (see p. 387).

Alimentary fever. Deviations from normal temperature curves occur with great regularity in the acute stage of acute gastroenteritis. The fact that subfebrile or more rarely febrile temperatures occur with acute diarrheal disturbances has been known for a long time. This fever was originally regarded as bacterial fever, until *Finkelstein* proved that the onset and the duration of temperature elevations were related to the food intake. An interruption in food intake brings the temperature curve back to normal. These correlations between nutrition and fever appeared so close that the temperature elevations were described as "alimentary fever."

The occurrence of this fever is, similar to the weight decrease, most closely connected with the water metabolism. Water deficiency at a certain stage produces temperature elevations independent of any food intake (see chapter on water deficiency, p. 251). An advanced state of thirst is accompanied in almost all children below one year of age by temperature elevations. Such observations can frequently be made in children with complete lack of appetite or suffering from violent vomiting. But not only an absolute state of thirst causes temperature elevations. Also with a relative water deficiency the body temperature rises. Whether a relatively small water intake produces elevations in body temperature depends on the type of other nutritional substances which are given together with the water. The various foodstuffs react differently with regard to their influence upon the water metabolism.

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[*] Values are given today usually in milliequivalents (1 milliequivalent = mg% x 10 x valence of element: atomic weight).

The greatest demands on water deposits in the organism are made by the protein in its metabolic actions in the body of the child. The salts are in second place, followed by the different types of sugar which probably, however, produce temperature elevations only indirectly, for instance through a diarrhea and the water losses connected with it. No increased demands on the water metabolism are known for fats.

Sudden increase of proteins in the diet or rapid intake of larger quantities of salts produces fever with great regularity, particularly in children during the first three months of life. This fever fails to occur when the increased supply of protein or salts is accompanied by a simultaneous increase in water intake. Similarly, temperature elevations will occur when the amount of protein and salt intake remains unchanged but the amount of water intake is relatively decreased, as happens in practice when concentrated feeding mixtures are given.

The alimentary fever can be treated in two ways: either by a reduction of the protein and salt quantities contained in the diet, if necessary discontinuing these substances altogether for a short period of time, or by increasing the fluid intake without changing the composition of the diet. Any so-called alimentary fever is, in the final analysis, a fever caused by thirst.

Two explanations have been brought forth for the development of fever due to thirst: The first theory claims that a disturbance in the physical heat regulation causes the "hyperthermia." The development of hyperthermia with a diet containing much protein and little water occurs as a result of increased heat production in the digestion of protein (specific dynamic action of protein). The second theory assumes that in the course of decomposition of the protein or perhaps also as a result of damage to the body cells, toxic substances develop in the circulation which irritate the centers of heat regulation. A deficiency of water which is necessary for further metabolism or elimination of these substances promotes the development of fever.

It is hardly possible to provide a definite answer to the question concerning the cause of the appearance of general symptoms. The general symptoms connected with acute gastro-enteritis, such as pallor, loss of turgor, hypertonia and hypotonia are a direct result of disturbances in water and salt metabolism and in circulation and of central nervous irritations or depressions. They are part of the symptoms which are today described under the chapter of neuropathology as disturbances of the vegetative regulatory mechanisms.

Pathologico-anatomical investigations have not succeeded in reducing the gaps in the theories as to pathogenesis of acute diarrheal disturbances. Hardly anything has remained from the original assumption which regarded acute dyspepsia as a disease of the intestinal wall for which a number of histological changes had been described. The finding of a "catarrh" of the intestinal wall as observed in post mortem examinations of children who had died of acute diarrheal diseases does not suffice to serve as basis for the pathological state. Even the occasional findings of more or less severe destructive and inflammatory changes in the intestinal wall are not enough to explain the abundance of pathological symptoms as observed in acute gastro-enteritis. In this respect the regularly observed findings of fatty degeneration and loss of glycogen in the liver, which might correspond to some disturbance in the liver function, seem of greater importance.

Only occasionally do we observe in severe acute gastro-enteritis serious destructive changes in the small intestines. Necrotizing and ulcerating inflammations and extensive gas formation in the intestinal wall occur although it has not been possible to isolate individual bacteria (at times proteus and pyocyaneus) in the severely affected sections of the small intestines. It is certain that the bacteria which cause this destruction in the walls of the small intestines are not of the shigella and salmonella group.

The many new occurrences which take place when the disease progresses from the first relatively harmless stage to the severe toxic form of the disease and the urgent measures which are required with the onset of the severe "metabolic catastrophy" justify a differentiation in the presentation of the two syndromes. In addition, the physician, outside of hospital practice, will under normal hygienic conditions treat almost exclusively patients suffering from acute dyspepsia (or acute diarrhea). Severe and toxic gastro-enteritis (intoxication) is a relatively rare disease under fairly good hygienic conditions. After a toxic gastro-enteritis has developed hospitalization of the severely ill infants is necessary in most cases since the diagnostic and therapeutic measures which are needed for curing this serious disease can be carried out only in the set-up of a hospital. For the practicing physician and for the pediatrician the common symptoms of acute diarrhea (acute dyspepsia) are therefore of greater interest.

ACUTE DIARRHEA. GASTRO-ENTERITIS SIMPLEX. ACUTE DYSPEPSIA

Clinical Symptoms of Acute Diarrhea

The onset of acute diarrhea or of a simple gastro-enteritis starts abruptly from the preceding period of health. The stools which on the preceding day still appeared normal or only slightly increased and pasty, become liquid, greenish or yellowish in color, mucous or shreddy. They are eliminated five to seven times a day and more. Despite adequate nutrition a weight loss sets in, which in simple acute gastro-enteritis usually remains within moderate limits. The younger infant loses 100 to 200g, the older infant occasionally more in the course of a day. Simultaneously the appearance of the child undergoes changes. The rosy color of the skin disappears. The children appear pale, greyish or livid. The tonus of the musculature decreases. The muscles appear flaccid and the abdominal muscles yield to the pressure of the distended loops of the intestines. Meteorism develops. The skin becomes withered and loses its lustre and its smoothness. Raised skin folds disappear initially within a short time, but as the condition progresses it takes a longer time for the raised skin fold to disappear. In fat children the subcutaneous fat becomes more solid and more granular. The psychological behaviour of the children changes simultaneously with these physical changes. A child suffering from acute diarrhea is irritative, sullen or sad. It is probably a question of temperament which determines whether the changes in the psyche of the infant manifest themselves in form of restlessness and frequent crying or in form of apathy. It might also be the presence or absence of colic-like cramps which cause painful crying in some patients and unusual quietness in others as part of the symptoms. Diarrhea, weight loss, changes in the tonus of the musculature and the turgor of the skin and psychological disturbances are always present among the symptoms of acute diarrhea (simple gastro-enteritis).

These constant symptoms are occasionally, but not regularly, accompanied by further disturbances. Gastric symptoms such as vomiting and regurgitation or lack of appetite, which not infrequently is the earliest sign of the disease, as well as changes in the course of the temperature curve are frequently observed. The physiological monothermia of the healthy infant disappears. The temperature curve, measured two to three times a day, is no longer an almost straight line. The differences between morning and evening temperature increase. Temperature elevations occur. However, the temperature rises rarely above 38 to 38.5°, provided there are no infections accompanying the pathological processes. In the urine we find protein, occasional leukocytes and cylinders. In premature and weak children we occasionally observe a moderate glycosuria already at this stage. Leukocytosis exists in the blood, and neutrophilia and a slight shift to the left in the white blood cells.

The number of these symptoms is considerably influenced, in frequency of occurrence and above all in their severity, by the nutritional state of the child. The pathological symptoms which have so far been mentioned describe as a total the approximate picture of acute diarrhea in the eutrophic child. In the dystrophic child the weight losses and the loss of tonus are greater and the damage to the children more pronounced. Lack of appetite, vomiting and fever occur regularly. For a child in an atrophic condition an acute diarrhea is a serious disease. As would be expected with the only loosely bound structure of tissues of the atrophic child, the weight losses are considerable. The symptoms of dehydration are more severe, and what is not destroyed by the acute diarrhea is demolished in the atrophic child by secondary complications, particularly infectious processes which develop unhampered in the tissues which have suffered additional damage through the acute diarrhea.

Changes in skin, in the tonus of the musculature, fever as well as changes in the urine etc. support the theory that the cause of the pathological symptoms, already in acute diarrhea, is a disturbance in the water binding power of the tissues and the cells. If the water binding capacities are still intact, as in the eutrophic child, the extent of the pathological symptoms remains small. In the child in a bad nutritional state whose ability for water absorption is already damaged, all symptoms become more pronounced.

Disturbances in water and salt metabolism exert an essential influence only upon the intercellular tissues. The structure of the cells themselves and thus the biologically important intracellular fluid is not yet affected, as far as can be concluded from the symptoms of the disease. Severe functional disturbances are still absent. These conditions, whereby essentially extracellular fluid is drawn upon to meet the increased internal fluid requirements, lead to a state which is described as dehydration (exsiccosis simplex). The relative lack of involvement of the intracellular fluid and the protein reserves in the plasma differentiate the acute diarrhea from severe stages of diarrheal diseases (see p. 343).

The prognosis of acute diarrhea is nowadays favorable. It depends less on the severity with which the pathological symptoms appear than on the nutritional state and the age of the child. In the eutrophic child the prognosis is always good, provided that no errors are made in the treatment. Even in the dystrophic child an unfavorable outcome is only rare. In those cases, as in any atrophic infant, it is not the acute diarrhea itself which endangers the life of the patient. The atrophic and the severely dystrophic infant dies of a secondary infection, or the lability of his tissue structure is so severely affected by this new damage caused by the diarrheal disease that even with an apparently light onset weight losses which cannot be stopped despite all therapeutic progress terminate the life of the child. Eutrophic

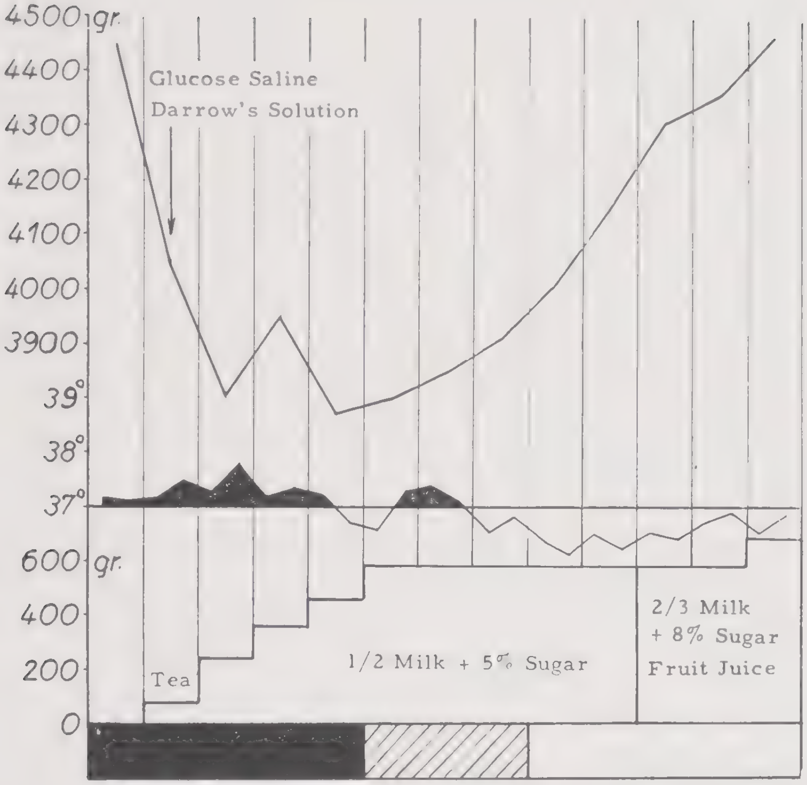

Fig. 33. Infant G.B., two months old. Acute diarrhea and acidosis. Cure after infusion of glucose-saline and Darrow's solution. Starvation period with tea only. Half milk and 5% sugar without sulfonamides or antibiotica.

infants during the first weeks of life react similar to atrophic children. At this early age any acute diarrhea signifies even today a serious disease (see also diarrhea of the newborn, p. 428).

The treatment of acute diarrhea has been greatly facilitated and improved due to progress made in the last years. In place of uncertain trials it is now possible to establish somewhat systematic indications for therapeutic procedures.

For the cure of acute diarrhea the following basic requirements must be met:

1) Treatment of diarrhea can and should take place in accordance with one of the recommended procedures. Although individual attention in the treatment of acute nutritional disturbances during infancy is indicated and even necessary in order to be successful it is important that less experienced practitioners

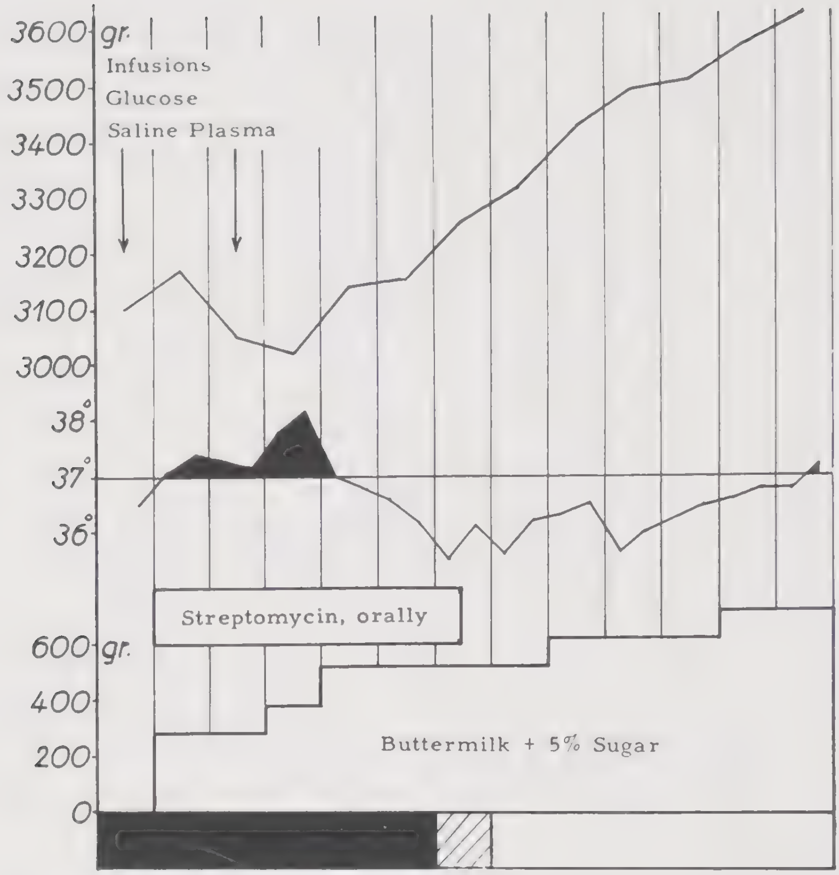

Fig. 34. Infant S.A., seven weeks old. Acute diarrhea with dehydration. Cure with diet of buttermilk and 5% sugar. Streptomycin per os.

follow some routine procedures for the light forms of acute diarrhea (acute dyspepsia). One method of treatment, with which the physician has obtained good results, should not without adequate reason be replaced by a new treatment procedure which is being reported.

2) The physician must have confidence in the method of therapy which he applies and must calmly await the outcome of his treatment. This advice for consistency is of importance because in the course of healing acute dyspepsia the diarrhea not infrequently persists for some time in undiminished force. The continuance of diarrhea frequently induces the physician to make repeated but superfluous and ineffective changes in his orders. Diarrhea alone, even if it continues to the third and fourth day of

the disease and even longer, must never be the cause for deviating from the method of treatment.

3) The aim in the treatment of acute diarrhea is to return, after as short a time as possible, to a diet which is normal and complete in quantity and composition. This aim is accomplished after an average of eight to ten days. Fear of an aggravation or a return of the diarrhea induces the less experienced practitioner not infrequently to misjudge the dangers existing in an excessively extended "therapeutic diet" or in a qualitative or quantitative hunger.

4) Dietetic therapy is even today the basis of any treatment of acute diarrhea. To this dietetic treatment drug therapy of diarrhea has been added in the last years as important supplementation.

There are a number of general rules concerning the feeding mixtures which are suitable for the treatment of acute dyspepsia. It seems necessary to mention them because so many different feeding mixtures have been recommended in the course of the past years for the treatment of acute diarrhea that they create confusion. Despite apparent differences and improvements the therapeutic diets, many of which are usable and have proved successful, can be traced back to a few basic principles. Mixtures which are composed approximately in accordance with the following outline are suitable for therapeutic food in acute diarrheal diseases:

1) The therapeutic diet should contain little fat.

2) An increase of protein in the diet is desirable. The practical advantage of protein rich mixtures in the treatment of acute diarrhea has been proved, independent of any theoretical interpretation.

3) Acidification of the food seems to be of advantage. Any acidification, whether produced artificially by addition of acids or by spontaneous acidification of milk (e.g., in form of buttermilk) has an anti-dyspeptic effect, provided it does not exceed certain limits.

The mechanism of the effect of acidity is as yet unexplained. It has not been proved and seems unlikely that the quantities of acid which upon intake of an acidified milk mixture pass from the stomach into the duodenum are so much greater than the acidification which develops with any type of food through the acid gastric juices and that these amounts of acid are capable of stopping bacterial growth in the small intestines. The more evenly distributed, fine and loose casein curd which develops in the stomach from any acidified food might facilitate gastric digestion. It might also be that the favorable effect of acidified therapeutic food is based on a much more complicated as yet unknown process.

4) Caution is necessary in calculating the carbohydrate quantities contained in the therapeutic diet. This caution must, however, not be carried to the point that the child is completely deprived of sugar, even for a few days. Even in a child with acute dyspepsia the quantities of sugar should amount to not less than 5% of the feeding mixture. The harmful effects of a sugarless diet upon the organism of the child must by all means be avoided. Caution in apportioning the carbohydrates refers to elimination of those types of sugars which are only slowly absorbed from the intestines and which produce increased fermentation in the intestines, such as lactose or malt extract. They are replaced by cane sugar, special sugar mixtures or still better by dextrine-maltose mixtures, the so-called "infant sugar." This applies similarly to the choice of second carbohydrates which are used for the preparation of diluting fluids in the therapeutic foods. Here too, the more rapidly absorbing and less fermenting types of flour such as rice flour, corn flour or similar types are preferable to those kinds of flour, such as oat and rye flour, which pass through the intestines more slowly and produce a stronger fermentation. The exclusive feeding of an infant with rice or oat water or similar food is in any case a mistake.

By observing these basic rules it is possible to summarize the older and newer more or less complicated feeding mixtures which are recommended for the treatment of acute diarrheal diseases. The physician can then choose among them on the basis of his experience.

1) Simple milk mixtures: 1/2 milk diluted with rice water, with the addition of 5% cane sugar or special sugar mixtures. These mixtures can be acidified by the addition of lactic acid, citric acid or hydrochloric acid.

2) Plain milk mixtures enriched with protein and sugar (5%), that is addition of 1 to 2% of a casein-calcium preparation (Casec etc.).

3) Half-skimmed acidified milk with the addition of rice water and 5% sugar.

4) Milk mixtures which are rich in protein, acidified and with relatively low fat content:

a) Buttermilk, which contains very little fat (1 to 1.5%), with 2 to 3% rice water and 5% special sugar mixtures or dextrine-maltose. In place of commercially available buttermilk, which frequently has too high a degree of acidity, canned buttermilk powder (Eledon etc.) is recommended, to which also 2 to 3% rice water and 5% sugar are added.

b) Protein milk (*Finkelstein-Meyer*) or one of the powdered preparations of protein milk. With this type of milk the not inconsiderable fat intake of 2 to 2.5% is tolerated without danger, even in acute diarrhea.

Protein milk is produced by finest dispersion of casein-calcium curds from 1 liter whole milk in half a liter buttermilk and replenished with water to 1 liter of the finished preparation. Protein milk contains per liter: 3% protein, 2 to 2.5% fat, 2% lactose, the whey salts from half a liter milk and the calcium salts from $1^1/_2$ liter milk. Protein milk is today universally available in powder form in commercially produced tins. The protein milk powder is diluted according to prescription in varying quantities of 10 to 12% in rice water, with 5% cane sugar or special sugar mixture added. Skimmed protein milk preparations are also commercially available.

The physician can choose from these types of milk mixtures depending on the age, the nutritional state or the amount of dehydration of the patient. Any of the above mixtures can be successful if the physician is familiar with them and adheres calmly to the chosen diet. In older infants and in lighter forms of the disease sweetened and possibly acidified half milk or $^2/_3$ milk will probably suffice. For severe forms of acute diarrhea the half-skimmed milk or protein milk would be indicated in eutrophic and dystrophic infants beyond the third month of life. For young infants during the first three months of life and for older children who had suffered considerable weight losses the buttermilk would be the diet of choice because of the greater whey content. But more important for the practicing physician than the utilization of one or the other type of therapeutic diet is the prescription of a feeding mixture with which he has had more experience.

Before starting with the therapeutic diet the sick child undergoes a certain period of starvation during which the child receives only tea or water. We cannot at present support recent recommendations to forego this period of hunger altogether and to begin immediately with smaller or even larger quantities of milk mixtures. The liquids which are given during this period of starvation must be sweetened in order to adapt them to the natural taste of the child. Saccharine seems more suitable than sugar for sweetening the weak tea or the boiled water. This certainly applies to the youngest infants. In older infants and in cases where the diarrhea is not too severe the water can also be given with special sugar mixtures or dextrine-maltose. Concentrations between 5 to 20% have been recommended for these mixtures. The physician must, however, be aware of the fact that the intake of carbohydrates creates conditions in the intestinal tract which promote the existence of bacteria. On the other hand the use of saccharine for sweetening the tea or water does not provide a medium for the growth of bacteria. The duration of this hunger period was formerly established more or less systematically at 12 to 24 hours in acute diarrhea. The possibility to rapidly attain today sterility of the small intestines through drug therapy permits a shortening of the period of hunger.

The duration of the period during which only tea is given is determined by three factors: 1) the age of the child, 2) the nutritional state of the child and 3) the severity of the disease.—ad 1) The younger the child, the less he can tolerate the period of hunger. The older child is much less affected by the interruption of food intake.—ad 2) While an eutrophic child, with his reserves of fat, glycogen and protein, can overcome even a longer period of starvation without considerable damage, any prolonged period of hunger represents a serious danger to the severely dystrophic and the atrophic infant who has no reserve substances

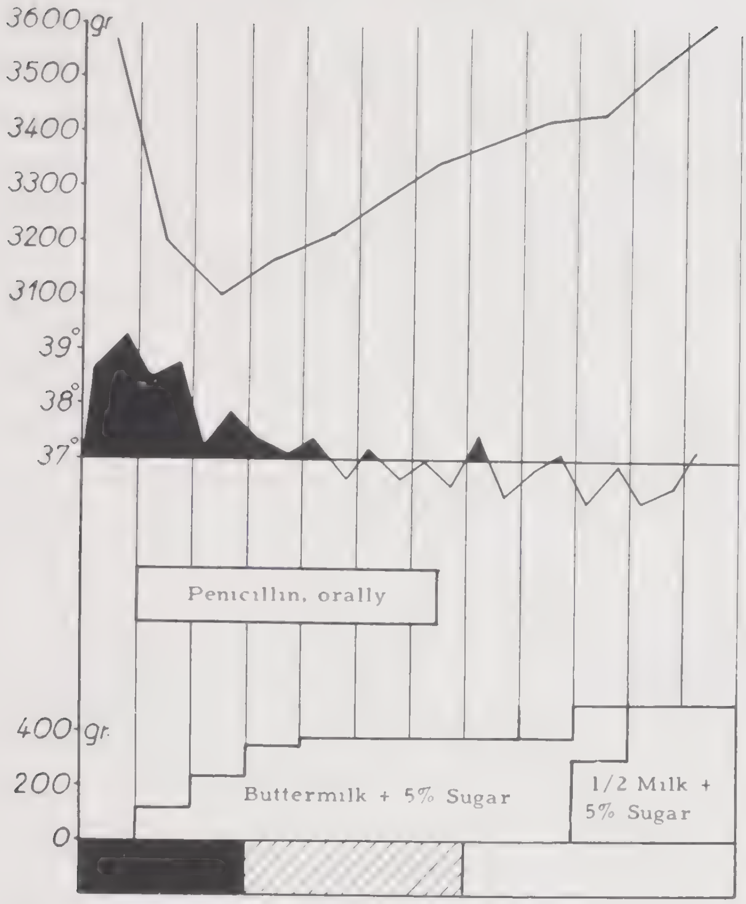

Fig. 35. Infant A.G., 15 days old. Acute diarrhea of the newborn. Cure with diet of buttermilk and 5% sugar. Penicillin per os (6 x 25,000 U.).

at all with which to maintain the most basic vital functions.—ad 3) In a less severe form of disease the hunger period can be shorter than in more serious cases. On the basis of these three factors the physician can determine in the individual patient the duration of the period during which only tea is given. In cases of simple acute diarrhea in the youngest children, in children with nutritional disturbances and in light forms of the disease this starvation period lasts about six hours. In older infants in eutrophic condition and with severe forms of the disease the interruption in food intake extends to about 8 to 12 hours. The possibility to more or less replace the enteral nutrition by parenteral supply of liquids, salts and nutritive substances (see p. 394) permits

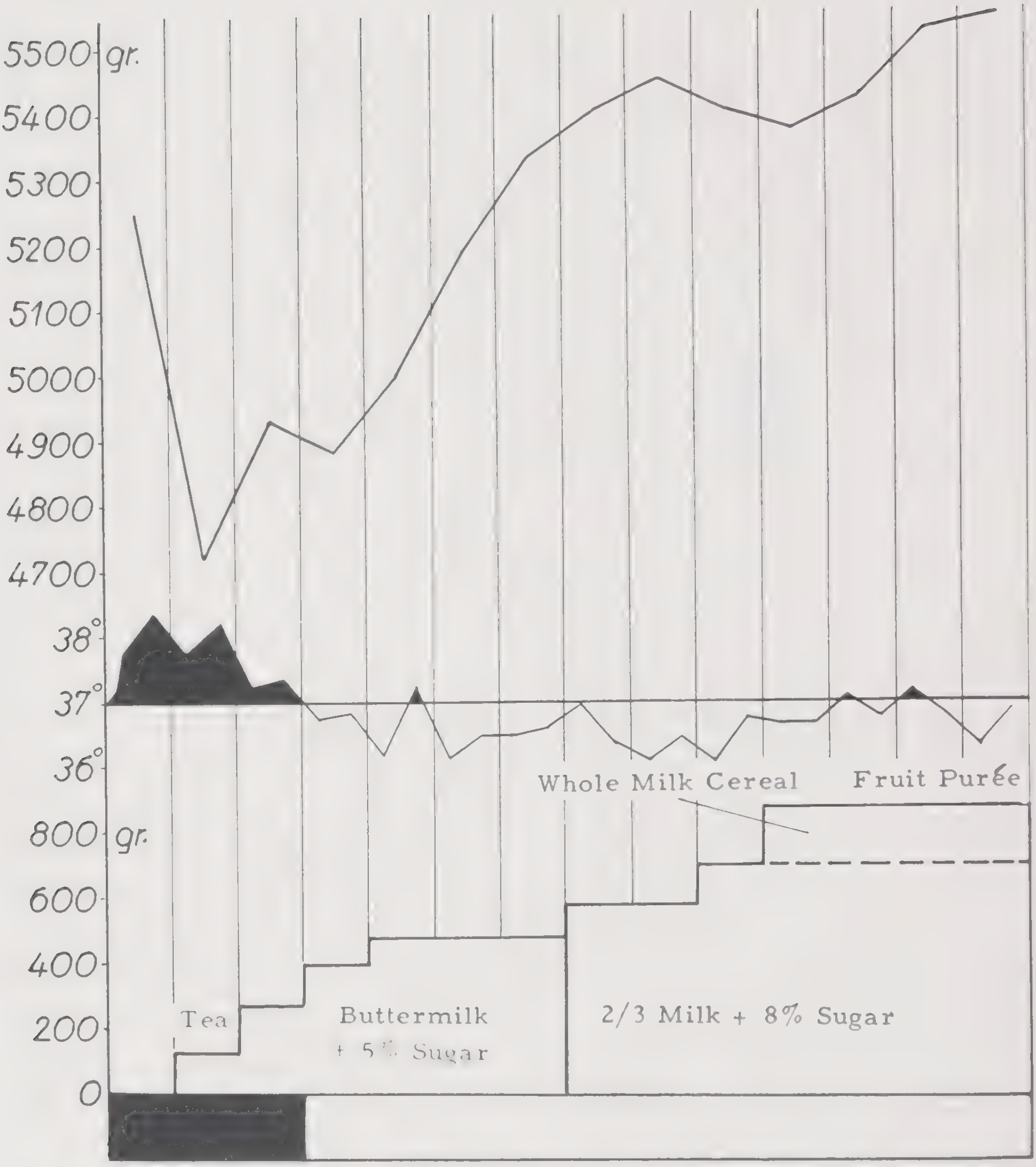

Fig. 36. Infant L.M., five months old. Acute diarrhea. Cure: starvation period with tea only. Buttermilk with 5% sugar *without* antibiotica or sulfonamides.

today an even further prolongation of the hunger period, if the physician considers this necessary. The need for a protracted parenteral fluid intake exists mainly in those forms of acute diarrhea which are also accompanied by gastric symptoms (vomiting) and thus make an adequate fluid intake per mouth impossible.

The resumption of oral feedings of such patients should be delayed until the more severe gastric symptoms (vomiting, nausea, lack of appetite) cease.

The effect of a hunger period upon the symptoms of acute diarrhea is impressive. The subfebrile temperatures disappear, the weight losses cease or continue in a greatly reduced measure. In fact we occasionally observe the paradoxical phenomenon that despite lack of food intake the weight curve begins to rise. Among the general symptoms the greyish color disappears very soon and the first rosy shimmer of the skin indicates the beginning of convalescence. The eyes regain their lustre and vitality. The tonus of the musculature and the turgor of the skin improve. Urine secretion again becomes normal. Appetite returns. Exceptions to these processes occur when parenteral or specific enteral infections were contributory to the development of the dyspepsia (see chapter on intestinal infections). In such patients the hunger period does not bring a critical drop in temperature, although the water diet not infrequently brings about a lowering of the temperature curve (see below). In hydrolabile children and in severely damaged dystrophic and atrophic children the period of hunger is not always followed by the hoped for cessation of weight loss. Instead we observe a further, at times even more abrupt weight drop which can be stopped only with difficulty and sometimes not at all. A number of young infants show similar reactions so that even an acute diarrhea becomes for them a serious disease.

With regard to the nutritional therapy which follows the period of starvation in acute diarrhea the procedure depends again on the nutritional state and the age of the child. As example for the dietetic treatment we shall choose the procedure followed in the case of acute diarrhea in an eutrophic child. The infant receives the following diet, distributed among five, six to eight meals, depending on the patient's appetite:

First day:	200–300g	of the mixture	= 5 × 40–60 g
Second day:	300–350g	” ” ”	= 5 × 60–70 g
Third day:	350–400g	” ” ”	= 5 × 70–80 g
Fourth day:	450–500g	” ” ”	= 5 × 90–100g
Fifth day:	500–600g	” ” ”	= 5 × 100–120g

The increase in food quantities takes place only after the results of the preceding measures can be observed. This requires 24 to 48 hours at the most. After this time it can always be ascertained whether the ingested food has improved or aggravated the curative process. In normally convalescing patients it is therefore permitted to increase the food quantities daily or every second day at the most, by about 100g. After one week the nutritional requirements of the child are usually covered.

The initial food quantities of 200 to 300g supply the child with about 120 to 180 calories. These small quantities meet the caloric requirements for the basal metabolism and thus prevent a further drawing from the child's own substances. An increase in food quantities is needed, provided that the general signs of disease disappear (see above), in order to avoid any further unnecessary hunger.

Any increase in food quantities is dependent on the course of the weight curve. This means that an increase in the food quantities is not necessary as long as the child shows an adequate weight gain (about 30g per day). The smallest food quantities with which the sick infant can rebuild his tissues are the optimal amounts. In this connection it appears surprising that infants, particularly those who had lost considerably in weight, begin after a while to show weight gains on quantities which are far below the calculated caloric requirements. This increase probably represents the re-establishment of the water and salt balance.

It usually takes somewhat longer for the diarrhea to cease although today, due to recent changes in drug therapy, pasty stools can be eliminated already on the third or fourth day of the disease. If the elimination of frequent, liquid, greenish and mucous stools persists for a longer period of time this diarrhea in itself, provided all other pathological symptoms improve, is never an indication to continue with a qualitatively and quantitatively inadequate diet and to delay further increases in food intake. An incorrect evaluation of the symptom of diarrhea—and this cannot be stressed too frequently—is a source of a number of dangers for the patient. It leads to the situation whereby a special diet, for instance only rice water or sweetened rice water or strongly diluted milk or calorically quite inadequate food quantities are continued for many days in order to "prevent irritation of the diseased intestinal tract." It even happens at times that a renewed reduction in food quantities or even a new period of starvation is ordered because of the elimination of soft stools. Any repetition of a period of starvation represents a dangerous procedure in infants, especially infants during the first months of life, particularly if the second or third hunger period closely follows the first one. A new period of starvation is unavoidable only in those cases where diarrhea is accompanied by a recurrence of general symptoms such as weight loss and signs of water losses or where gastric symptoms persist or reappear. The stools gradually resume their normal color and consistency, the mucous discharge ceases after the infant has been fed for a number of days and begins to gain weight. Normal stools frequently appear only when the diet becomes again adequate in quantity and composition.

The therapeutic diets for acute diarrhea are composed in such a way that they cure the diarrhea rapidly and partly, as for instance buttermilk, that they restore the water absorption. Therefore they are usually not qualitatively complete diets with regard to their composition. In the acute phase of the disease the need for a complete diet is not of such great importance. After the first acute stage of the disease, particularly after the diarrhea has improved, a rapid return to a complete diet is, however, desirable. This applies mainly to the intake of fat which in most therapeutic diets (for instance in buttermilk and in half-skimmed milk) is not contained in sufficient quantities or is lacking altogether. The addition of 1 to 2% butter or of a roasted butter and flour mixture or transition to a $^1/_2$ milk or $^2/_3$ milk is indicated and possible during the second week of the disease, provided convalescence proceeds normally.

The procedure which has just been outlined is indicated for the treatment of acute diarrhea in eutrophic infants during the first five to eight months of life. In older children the process can be accelerated. In these cases the nutritive substances from the mixed diet of the older child (white cheese, puréed meat, rice cereal, bananas), which have a constipating action, can be added to the diet at an early time.

The use of substances such as puréed apples or puréed bananas, which are rich in pectin, or carob flour which is rich in lignin, is

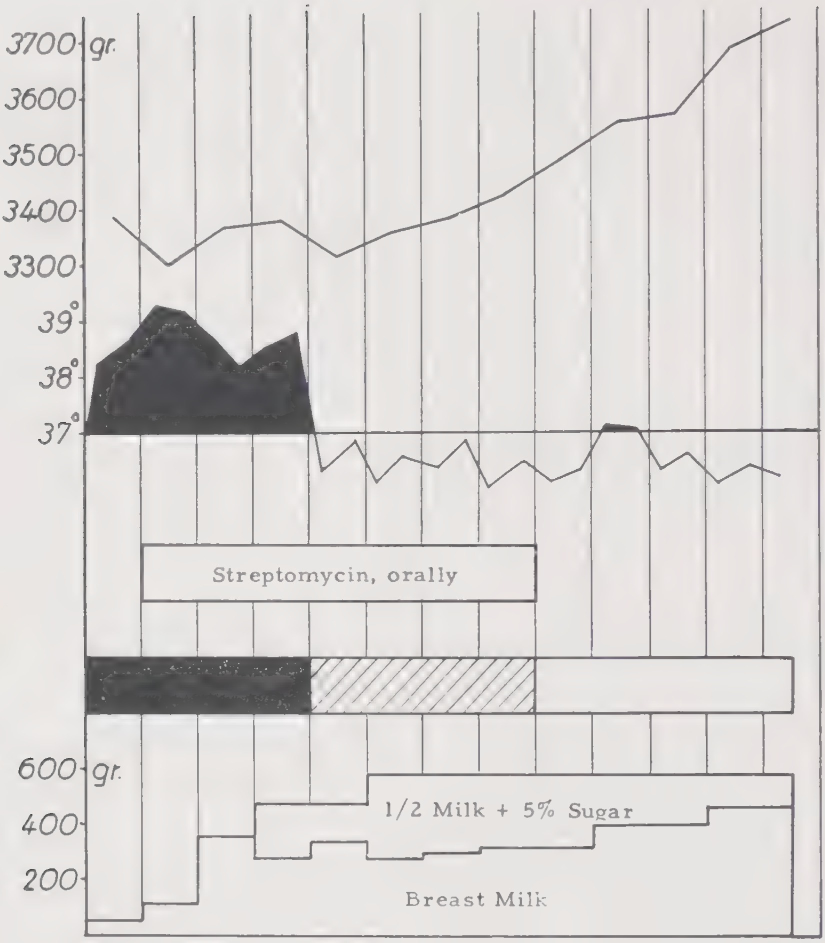

Fig. 37. Infant M.P.N., three months old. Acute enteritis on a mixed milk diet. Diarrhea, loss of weight, etc. on a diet of breast milk plus $^1/_2$ milk. Discontinuation of $^1/_2$ milk. Only breast milk for a few days, then again addition of $^1/_2$ milk. Streptomycin, orally.

indicated for young infants whose stools improve only slowly following an acute diarrhea, or for infants beyond the third month of life beginning from the onset of the disease. Many years ago already a carrot soup (*Moro*) was recommended for initiating the treatment of acute diarrhea.

500g fresh carrots are washed, cleaned and cut in pieces, and are cooked with 150 g water for 15 minutes, preferably in a pressure cooker. The vegetable is passed through a fine sieve and hot water is added to make up 1 liter. 3g cooking salt is added.

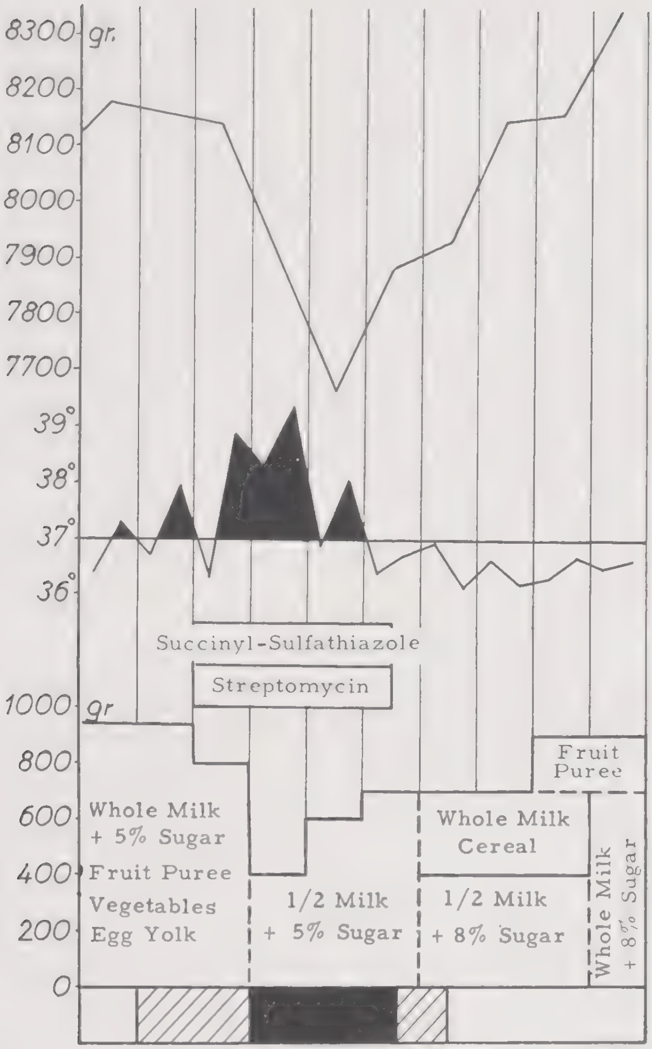

Fig. 38. Infant M.A., nine months old. Acute gastro-enteritis. Rapid cure with $^1/_2$ milk plus 5% sugar. Streptomycin per os (4 x 50 mg.) and succinylsulfathiazole (4 x 0.5g).

With these types of diet the transition to a normal diet occasionally presents difficulties since the stools, which had become more solid with the carrot or banana diet, again become more liquid and more frequent as soon as a milk feeding is added. A combination of fruit purées (carrots, bananas, carob flour, apples) with buttermilk can be given from the first day of disease to older infants with a not too severe form of acute diarrhea.

Carob flour which is today produced commercially (Arabon, Ceraton, etc.) from roasted and ground carobs (Ceratonia siliqua, French: caroube, English: locust bean, St. John's bread, Italian: Cairuba) has been found effective in the treatment of diarrhea.

> The powder is mixed well with boiling water and again brought to a boil. For young infants 1% carob flour is added to one of the chosen therapeutic diets ($^1/_2$ milk, buttermilk). Other authors recommend that treatment be initiated with a 5% flour mixture, without the addition of milk. To this an increasing quantity (5% at first, later up to 10%) of buttermilk powder or skimmed milk powder is added after two to three days. After the first day of treatment 5% sugar (special sugar mixture) is added to the mixture. The preparations of carob flour have also been found effective during the period of convalescence from the acute disease when the stools maintain the pasty or mucous appearance for a longer time.

Although the treatment of acute diarrhea is relatively easy and simple in the eutrophic child, the cure of the same disease presents considerable difficulties in dystrophic or atrophic infants. The basic danger in those cases is not the diarrhea. In these nutritionally damaged children the diarrhea can never be cured with the certainty which is the case in eutrophic children. This delay in the cure of the diarrhea leads to a situation whereby the child is kept for a longer period of time, or is put back repeatedly, on a special diet which further damages the organism. In acute diarrhea of dystrophic and atrophic children the therapeutic procedures are dominated by the desire to stop the diarrhea. Therefore, for fear of a recurrence of the diarrhea the transition to the necessary quantitatively and qualitatively complete diet is delayed. In order to shorten the period of hunger as far as possible the mixtures chosen in the treatment of acute diarrhea in nutritionally damaged children should be those which regulate the pathological intestinal processes rapidly and with certainty. Half-skimmed acidified milk in cans, buttermilk with 5% sugar and 1–2% rice water, and protein milk with 5% sugar are indicated for those patients.

Breast milk is not the ideal food for infants suffering from acute diarrhea, particularly for dystrophic and atrophic infants during the acute stage of the disease. Its ability to restore the functions of the damaged cells is unequaled by any other type of food. On the other hand, its effect in the treatment of diarrhea is negligible. Because of its low salt and protein content breast milk is, furthermore, not very suitable in replacing those substances which were lost and which are required for the restoration of the tissues. The results of exclusive breast milk feeding in acute diarrhea are therefore frequently a continuation of the diarrhea and absence of weight gain or even further weight losses. For this reason it seems safer, particularly in children whose nutritional state is below normal, to begin the cure of acute diarrhea with a therapeutic diet, for instance sweetened buttermilk, and only after the acute condition has improved, to add breast milk to the therapeutic diet or, in the youngest infants, replace this diet completely with breast milk. Under no circumstances, however, may breast milk, if given as the only food in the treatment of acute diarrhea, be given in different quantities than any other therapeutic diet. In these patients, too, the addition of small quantities of a salt and protein rich food is necessary after a few days in order to supplement the breast milk, similar to the procedures recommended for the nutrition of premature infants (see p. 171).

In all children beyond the third month of life who suffer from acute diarrhea, vitamins (fruit juices etc.) must be supplied at an early date (see p. 153) or one of the vitamin C or B containing preparations (tablets, solutions) should be given. This additional intake is permitted at an early time, even if the stools are not yet "normal." An aggravation of the diarrhea due to the addition of small quantities of fruit juices (30–60g daily) should not be feared.

Until about ten years ago there was de facto no drug therapy for the treatment of acute diarrhea. The treatment given in infant wards of hospitals was predominantly dietetic. So-called "constipating medicines" were not employed at all. It was, in fact, understood that the pediatrician would treat the disease without drugs. In the private home such an attitude was not always possible. In view of the frequent, liquid, semi-liquid or mucous stools the physician was forced to make an attempt to bring the stools back to "normal" with the aid of drugs. Such a procedure proved effective only in cases of dyspepsia which were already in the process of being cured. In the first stage of acute diarrhea the prescribing of any of these drugs was ineffectual. In the convalescent stage of diarrhea, when loose stools are left as the only remaining symptom, so-called adstringents and adsorbents occasionally improved the consistency of the stools. The following were recommended: Calcium in form of calcium carbonate, Bismuth in form of bismuth subgallicum (Dermatol) (five times $^1/_4$ teaspoon), bolus alba sterilisata (three to four times $^1/_2$ teaspoon), tannin preparations and animal charcoal. All these preparations are effective essentially in the large intestines. The stools then become at times less frequent and more solid. Altogether the results obtained with these treatments were at least doubtful. The medications are never successful in the acute stage of diarrhea. Prescribing these drugs without ordering changes in the diet is a mistake. A warning must be given against ordering castor oil or calomel at the beginning of an acute diarrheal disease.

Revolutionary changes in the drug therapy of acute diarrheal diseases occurred only in the last years by the introduction of sulfonamide preparations and shortly thereafter of antibiotica into the therapeutic armamentarium. The application of these drugs brought basic changes in the therapy and in the process of healing of the diseases. The duration of the disease was shortened. The prognosis of even severe forms of the disease improved radically. Unfortunately it is not as yet possible to give clear indications as to which of the many applicable preparations are indicated for the individual case. Besides, these therapeutic measures still undergo constant changes due to the introduction of new preparations. Apparently the degree of efficacy of one preparation or another depends on the type of bacteria which participate in the development of the disease as their sensitivity toward the different drugs varies. At present the physician is still more or less dependent on his own experience or on a trial and error experimentation. Chemotherapy with sulfonamide preparations and with antibiotica has scored its most impressive successes in the severe forms of acute diarrheal diseases, the acute toxic gastro-enteritides. In the lighter cases of acute diarrhea, dietetic and drug therapy appear today about equally effective. The relatively light forms of the disease, particularly in eutrophic children, should even today be treated exclusively with dietetic measures. It appears important to stress this since some of the drugs (sulfa preparation) are not always harmless and others (e.g., streptomycin) cause apparently a rapid adaptation and resistance on the part of the bacteria against these drugs; resistant strains are not inhibited in their development or destroyed. Despite this the description of the therapy with these drugs should be included in the discussion of treatment of acute diarrhea since it has become customary to prescribe them in every case of acute diarrheal disturbance and their use is undoubtedly indicated in acute diarrheas of dystrophic and atrophic children.

Sulfonamide preparations, including sulfathiazole and sulfadiazine, seem to be adequate for the treatment of many acute diarrheas. These preparations are prescribed in amounts of 0.1 to 0.2g per kilogram of body weight per day. The calculated total quantity of the drug is distributed among four to six portions which are given at regular intervals throughout day and night. In some children these drugs may cause vomiting, and in such cases the drug should not be given but replaced by other medicaments (see later). The medication of sulfa preparations should be continued until the stools are approximately normal and the child begins to gain weight. Because of the danger of possible damage to the blood (anemia, agranulocytosis) and the kidneys (erythrocyturia and anuria) the blood picture must be checked every other day, particularly the number of leukocytes, and the urine must be examined microscopically. A rapid drop of leukocytes to values below normal necessitates immediate discontinuation of sulfa therapy. The appearance of larger numbers of erythrocytes in the urine is a contra-indication for the continuation of treatment because of the danger of anuria through obstruction of the urinary passages with sulfa crystals. The appearance of sulfa crystals alone in the urine permits continuation of the treatment. The slowly absorbing sulfa preparations which in part reach the lowest sections of the intestines, such as sulfaguanidine, succinylsulfathiazole, phtalylsulfathiazole, etc., can be used in non-specific diarrheas in which coli bacilli or paracoli bacilli were found in the stools. The slowly absorbing sulfa preparations must be given in larger quantities (0.2 to 0.3g per kilogram of body weight per day during the first days, then gradually less). During treatment with sulfa preparations reddish-violet masses occasionally appear in the vomitus and the stools, which may be erroneously interpreted as blood admixtures. They are in fact reddish colored products which develop from the sulfa preparations in the gastro-intestinal tract. Their appearance is harmless and should not prevent continuation of treatment. Certain races, for instance inhabitants of the Eastern Mediterranean and of North Africa, seem to be particularly sensitive against sulfa preparations. It is always necessary to insist on adequate fluid intake during sulfa therapy.

Among the antibiotica the oral administration of penicillin, streptomycin, aureomycin and chloromycetin has been recommended. The efficacy of penicillin seems uncertain. This drug has been found effective only in a few epidemics of acute diarrhea, given in quantities of six to eight times 10,000 to 25,000 units daily per os. Better results have been obtained with oral administration of streptomycin (in quantities of 50 to 100 mg, four to five times daily). The oral administration of streptomycin seems particularly indicated in acute diarrheal diseases in the youngest infants. A combination of one of the slowly absorbing sulfa preparations together with streptomycin is at present perhaps the most effective drug therapy of acute diarrhea. Chloromycetin is effective in specific infections of the intestinal tract (see later) as well as in most acute diarrheas, even if the etiology of the disease is not clear (see diarrhea of the newborn). Chloromycetin is given in quantities of 30–50 mg per kilogram of body weight per day, divided into four portions. Despite its bad taste chloromycetin—and the same applies to aureomycin—is taken by most infants without difficulties. The drugs only rarely cause vomiting, even in the younger infants. Aureomycin occasionally causes an aggravation of the diarrhea at the beginning of the treatment. In spite of this the drug is effective in non-specific and specific intestinal infections (in quantities of 30 mg per kilogram of body weight per day). The increase in appetite which has been reported has not been observed by us. Chloromycetin and aureomycin seem particularly indicated in cases where the diarrheal disturbance was caused by a parenteral infection (otitis, pneumonia) or where secondary infections developed in the course of the disease.

The medication of these drugs, particularly the slowly absorbing sulfa preparations, the streptomycin and the chloromycetin, results in a more or less complete sterility of the intestines. The bacterial life in the small and large intestines comes to a standstill, a condition which *Freudenberg* described as "amicrobiosis intestini." The stools of children in such a condition then show a neutral reaction. If the absence of bacteria in the intestinal tract persists over a longer period of time, that is if the medication, particularly of streptomycin, is continued for too long, a tendency to bleeding may develop. The cause for this is a disturbance in the formation of vitamin K in which the intestinal bacteria normally participate. It is, however, not possible to prove in all patients prolongation of the prothrombin time. In any case the obstruction in the development of bacteria in the intestinal tract should not be continued over an excessively long period of time. The medication of streptomycin and other preparations which also "sterilize" the large intestines should be interrupted after eight to 10 days at the most. Penicillin and sulfa preparations which are absorbed in the small intestines are less limited as to the period of administration. There is a further danger that other germs and fungi which are resistant to antibiotics settle in the intestinal tract which has become practically free of germs due to suppression of the physiological flora. There have been reports of infections with staphylococci which are resistant to streptomycin and of severe infections with monilia albicans.

The successes which have been obtained with the introduction of sulfa preparations and of antibiotica in the treatment of acute diarrheal diseases and particularly in the toxic forms of the disease (gastro-enteritis toxica gravis, see later), are considerable. The decrease in mortality from this disease and the considerable shortening of the period necessary to cure even severe cases is surprising. This progress in drug therapy is probably the reason why after the introduction of these drugs there have been hardly any reports in the last ten years regarding essential innovations and improvements in the dietetic therapy of acute diarrheal diseases.

In the course of the first hours and days of acute diarrhea severe complications can arise, particularly in children whose nutritional state had already suffered damage, which require immediate attention. This includes the failure of the peripheral circulation which in dystrophic and atrophic children causes an acute state of collapse. Symptoms pointing toward this danger are pallor of the child, restlessness, reduction in urine secretion, slowing down of heart action with the heart tones becoming softer and duller, and enlargement of the liver caused by collection of blood in the intestinal organs. In order to improve these conditions the administration of rapidly acting stimulants is indicated which mainly relieve the collapse of the circulation.

The formerly customary medication of camphor oil and caffein has been abandoned. Their place has been taken by intramuscular injections of cardiazol (2–5 tenth of a ml, depending on the age of the child, every three to four hours or more); injections of adrenalin ($^{1}/_{10}$ to $^{3}/_{10}$ of a ml, depending on age, of a 1:1000 solution i.m. every two to three hours) until the danger of collapse is removed. Warm baths (37 to 39° C) and hot water bottles are necessary for all children whose extremities, forehead and nose appear cold as signs of a failure of the circulation. The injection of cardiazol not infrequently produces tonic-clonic cramps which then in turn require injections in adequate quantities of anti-convulsive drugs (sodium luminal, Sevenal). Appropriate dosages are about: 0.03g sodium luminal for infants of the first three months of life, 0.05g sodium luminal for infants of three to six months, and 0.1g sodium luminal in older infants.

In cases of violent vomiting a lavage might occasionally be indicated in addition to the interruption in food intake per mouth. The important part then is not to rinse the stomach with large quantities of liquid until the rinsing fluid comes back clear; more important and much less tiring for the sick child is a single complete filling of the stomach with 100 to 150g of a slightly alkaline mineral water which is left in the stomach.

An important aid in the therapy of acute diarrhea is the nursing care of the child. The care of an infant suffering from an acute diarrheal disturbance is difficult. The bad mood and the negativistic tendencies of the patient make it still harder to care for him. Playing, talking, singing and similar entertainments which please the young infant find no response in the child suffering from dyspepsia. It is therefore more important that mother and nurse avoid everything which might increase the discomfort of the child. Regularity in food intake and regular periods of sleeping and waking should be maintained as far as possible. This includes the provision for undisturbed sleep. Unnecessary lighting of the room during the night should be avoided. Infants who usually have a light sleep would be given a sedative (luminal, medinal, adalin, etc.) in order to insure at least a few hours of sleep at night. Under such conditions the infant will feel better and will more easily overcome the disease than if the child is left restless and discontent. The experienced nurse will not only observe most carefully the instructions given by the physician regarding number and distribution, quantity and composition of the individual meals, but she should also be prevailed upon to report to the physician her observations of the sick child which will give him indications as to further therapeutic procedures. This includes exact recording of weight, stool eliminations, fever, etc., also in the treatment of an infant in the private home. It seems of importance to mention these requirements since the nurse who observes the patient all the time can form a better judgment regarding the course of the disease than the physician who only sees the patient for a short time. The experienced physician will therefore not find it below his dignity to be guided by the information of an able mother or nurse.

In the physical care of a child with acute dyspepsia particular care must be taken of the skin, from the beginning of the disease. Even children who ordinarily show no tendency to skin irritations are threatened with intertrigo due to the frequent eliminations. Frequent changing of diapers, careful cleansing of the soiled skin with oil or paraffin oil—not with water—covering of the most endangered areas on buttocks and upper thighs with gauze, with zinc oil, zinc paste, or powder, will reduce the danger of skin irritation which aggravates the restlessness of the child. Intertrigo can be treated by covering the affected areas with balsam of Peru or with a gentian violet solution. Older infants suffering from severe intertrigo, in whom the danger of being chilled is less great, can lie for hours without diapers, with warmth supplied by a heat lamp, an electric light cradle or similar apparatus. Even during the acute stage of the disease the daily bath must not be omitted. Adequate heat supply is important for every child suffering from diarrhea. Care should be taken that the room temperature should not drop too much at night. In children with a tendency to sub-temperatures, which are apt to occur particularly in dyspeptic atrophic children during the period of low food intake, a drop in the body temperature should be avoided by placing frequently changed hot water bottles around the patient. At times it might be necessary to place emaciated atrophic children in an incubator. On the other hand too much warmth and too much clothing might be harmful to the child, particularly in the hot summer months. The feeding of the infant requires special care. It becomes particularly difficult when there is a tendency to vomiting. The food must then be given slowly, with frequent and short interruptions. In place of a bottle it might at times be preferable to feed the infant with a spoon. Other children might drink better when the food is only slightly warmed or even cold. In order to avoid the swallowing of air children should always be fed in a half-sitting position, never lying down.

During the days when only small quantities of food are given particular care must be taken that the fluid requirements are covered. In order to satisfy the thirst it is sufficient to give dyspeptic children small quantities of tea sweetened with saccharine after or between meals. With oral fluid supply it is usually possible in children with acute diarrhea to obtain a fluid intake of about 150g per kilogram of body weight. Fluid intake per mouth seems to be superior to all other ways with regard to the assimilation of the ingested fluid. The generally low degree of dehydration in acute dyspepsia does not necessitate parenteral fluid supply in the majority of patients. Only in cases of acute diarrhea in severely dystrophic or atrophic children and in acute states of dehydration which might develop as a result of frequent vomiting and overheating it might become necessary to also supply fluids parenterally (see later).

Despite adequate treatment the course of acute diarrhea does not always proceed as smoothly as has so far been presented. It is of importance to know the correct procedure in treatment of a number of difficulties which may occur. These disturbances in the process of healing occur only rarely in the care of the individual child. In institutional care they are frequently caused by secondary enteral and parenteral infections. The most frequent disturbances in the course of treatment are the following:

1) The event which can most easily give rise to therapeutic errors is the continued presence of diarrhea. This has already been mentioned. If all the other general symptoms of acute diarrhea disappear, particularly if weight losses have already stopped or the weight has begun to increase, the elimination of frequent stools must never be the cause for deviating from the once started pattern of treatment of dyspepsia. With continued increase in food intake the ensuing improvement in the nutritional state also brings the diarrhea to a stop. The addition of carob flour (Arobon etc.) to the food, in quantities of 1 to 2%, has a "beautifying" effect on the frequency, consistency and color of the stools.

2) The absence of any considerable drop in temperature following the period of exclusive tea feeding may be disregarded in the therapeutic program provided the other symptoms of acute diarrhea (particularly pallor and loss of weight) show an improvement. These increases in temperature during the first days of treatment of dyspepsia can usually be traced back to parenteral infections (grippe, pyuria, otitis) which could either have caused the acute diarrhea or have developed in the organism which had been weakened through the acute disease. In the eutrophic and slightly dystrophic child with his good immunity these diseases are usually overcome without difficulty. In severely dystrophic and atrophic infants primary or secondary infections frequently occur in severe forms. Until recently they were not infrequently the cause for an unfavorable outcome of many cases of dyspepsia in atrophic children. With the introduction of sulfa preparations and antibiotica secondary infections have become less frequent, with the result that the prognosis of the disease has been improved considerably.

3) In dyspeptic children who had undergone a period of starvation or had been fed with only small quantities of food the turning of the weight curve to a stop in weight loss or to a weight gain does not always occur as promptly as described in the outline. Particularly in hydrolabile children and in those who before the development of dyspepsia had received a diet rich in

whey and sugar the first larger weight losses, brought about by the starvation period and the period of calorically insufficient food intake, are followed by further decreases in weight which, however, get progressively smaller so that the gradually decreasing weight curve soon begins to show a parabolic upward trend. These further weight decreases with a gradually turning weight curve are also no reason for deviating from the pattern of treatment, provided that the general condition of the child shows simultaneously an improvement.

4) Weight losses which persist at an undiminished rate or even at an increased rate are of entirely different significance. This transition from moderate weight decreases of acute diarrhea to abrupt weight losses which are not part of the symptoms of acute diarrhea are accompanied by a further aggravation in the general condition. Increased pallor, further loss of turgor, stronger diarrhea and frequently renewed temperature rises are the most striking characteristics of continued deterioration. A recurrence of the acute diarrhea develops in this manner which can deteriorate into a toxic gastro-enteritis (intoxication, see later). This turn to the worse develops particularly in those acute diarrheas which were caused by an acute infection, or it occurs when the acute diarrhea takes place in a non-resistant organism, e.g., a premature or an infant who had been weakened through other preceding diseases. In severely dystrophic or atrophic infants whose cell structures had not yet been completely restored this unfavorable course of acute diarrhea is not infrequently observed. Such conditions have also been described as hidden decomposition (*Finkelstein*). In those patients, even if they are in a considerably progressed state of recovery, an acute diarrhea can, despite adequate treatment, turn inexorably into a severe diarrheal disease.

5) Similar complications occur occasionally also in a later stage of recovery from diarrhea, at a time when the symptoms of acute diarrhea had already disappeared. These renewed weight losses with recurring fever and diarrhea take place if infections occur around the first or second week of the disease or if, with rapid cure of the diarrhea and rapid onset of weight gain, the food quantities are, contrary to the routine, increased too rapidly or if the composition of the diet is changed too quickly, e.g., the fat

content increased too rapidly. Here, too, children in a bad nutritional state are particularly endangered. The treatment of this second disturbance should include, depending on the severity of the new attack, a repetition of the therapy applied to acute dyspepsia or toxic gastro-enteritis, supplemented with parenteral fluid intake, dietetic and drug therapy (antibiotica, sulfa preparations). The dangers of such a second starvation treatment so shortly after the first one are always great, particularly in cases where the first attack was severe, where the patients are young infants during the first three months of life, where the patient is in a bad nutritional state or where the second disturbance occurs in form of a severe diarrheal disease. Here, too, adequate application of infusion therapy has considerably improved the outlook.

6) Some dyspeptic patients who in the beginning apparently recover satisfactorily, after two to four weeks undergo not infrequently a period of poor development with recurrent diarrhea and slight temperature elevations. This disturbance in the process of recovery from the disease usually occurs at the time when the child has regained the initial weight losses but has not yet exceeded them. The organism has not yet regained the ability to build new cells at a normal rate. This dyspeptic-dystrophic stage can persist for one to two weeks or longer, despite calorically adequate food intake. A repetition of the starvation diet is not necessary in these cases. Some of the children, after a few days of standstill in weight gain spontaneously begin to show a rise in the weight curve. This also brings the diarrhea to a stop after a short time. For other children the addition of small quantities of protein rich food or the increase in sugar intake can be recommended in order to bring about a weight increase. Occasionally the absence of weight gain is the result of a vitamin deficiency. Addition of fruit juices, of half an egg yolk or of vitamin C and B containing preparations are indicated in such cases.

TOXIC GASTRO-ENTERITIS

(Toxicosis, Alimentary Intoxication)

The term intoxication or toxic gastro-enteritis has been chosen for this syndrome with good reason. In many respects the picture resembles a state of poisoning. The pathological processes on the part of the gastro-intestinal tract, particularly the loose stools which characterize the acute diarrhea, become of less significance compared to the other alarming symptoms, especially at the onset of the disease. The therapeutic measures during the first hours and days of the disease are determined by the state of intoxication and its consequences and not by the diarrhea.

The changes in the symptomatology, the development of symptoms which are lacking in acute diarrhea, produce a pathological picture which requires separate presentation.

However, there is no definite dividing line between acute diarrhea (dyspepsia acuta) and toxic gastro-enteritis (intoxication). In some cases it might be doubtful whether the disease belongs still to one or already to the other form of acute diarrheal diseases. In such patients it is customary to speak of pretoxic or subtoxic conditions.

The fact that intoxication (toxic gastro-enteritis) does not appear suddenly proves the connection between the two syndromes. The toxic condition is almost always preceded by a dyspeptic state which develops into an intoxication if the initial stage of diarrhea is not recognized, not sufficiently attended or inadequately treated.

Today toxic gastro-enteritis has become a rare disease while acute diarrhea continues in hardly diminished frequency. Under good hygienic conditions and with appropriate nutrition an intoxication develops only when the child is afflicted with an enteral infection or more rarely with severe parenteral infections. Avoidance of hunger, prevention of dystrophy or atrophy through a rational complete diet, the strengthening of the total resistance which is connected with it, contribute to the considerable decrease in the number of cases with severe gastro-enteritis (alimentary intoxication).

A neglect in the prophylactic measures even for a short time, a deterioration of the milk supply and milk hygiene, or a lowering of the living standard to unfavorable hygienic conditions suffice, however, to bring about an accumulation of severe forms of acute diarrheal diseases. These facts have been borne out by the events of the last war, the experiences of crowding together large numbers of children under unfavorable hygienic camp conditions and the migrations of large masses of population. A decrease in the occurrence of severe forms of acute diarrheal disturbances has been observed only in those infants living under the fortunate set-up of well planned hygienic conditions under constant medical supervision and under a developed system of preventive health care.

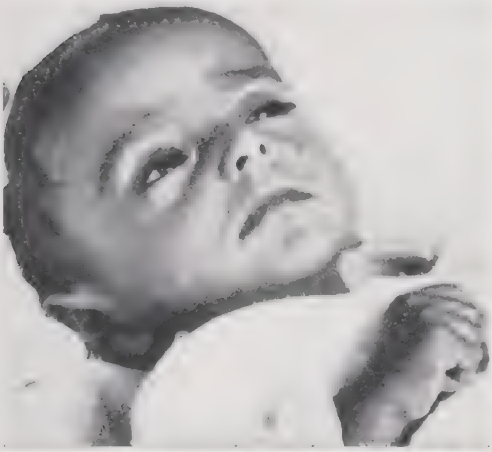

Fig. 39. Infant with toxic gastroenteritis.

The state of dyspepsia which precedes any alimentary intoxication shows no special traits which would characterize it as precursor of serious threatening danger. At this time the clinical diagnosis can only be that of acute diarrhea. During this preparatory stage, with diarrhea and occasional gastric symptoms dominating the picture, there are as yet no signs of a state of intoxication. The children are restless, sleep is disturbed, the appetite diminished; a pallor which can be better observed in the sleeping child than in the waking child will upon careful investigation point to a disturbance in the general condition. Particularly in the youngest children, in dystrophic and atrophic infants the severe disease develops suddenly, at times in the course of a few hours. Occasionally the introductory phase of the disease can be so short that the severe toxic gastroenteritis appears almost as the first syndrome. This precipitated course occurs particularly in the youngest infants and in cases of severe damages such as overheating on a hot summer day or a toxin poisoning in the course of acute shigella of salmonella infections.

The transition from the relative calmness of the preparatory diarrhea to the alarming events of fully developed intoxication is characterized by the appearance of special symptoms. Under signs of a collapse the child reaches a degree of irritability which can be aggravated to a state of tonic-clonic convulsions. A suspicion of a meningeal disease or of encephalitis now appears, particularly since the body temperature in some of the patients shows considerable increases at this stage. The period of restlessness, of symptoms of motoric irritability of encephalitic character accompanied by a monotonous crying or by convulsions, usually does not last long. The child soon falls into a state of apathy, followed shortly by a precomatose condition which may develop into coma. The symmetry of movements of arms and legs, which is typical for the healthy infant, disappears. The cause of these changes are disturbances in the central nervous system. The child executes motions which for instance greatly resemble the frequently mentioned so-called fencing position or the positional reflexes. At this moment the picture resembles in fact a state of severe poisoning. It is not always easy to recognize the dimming of consciousness, particularly in the youngest infants whose emotional reactions are rather limited even in days of health. Children suffering from toxicosis can be awakened from sleep only with difficulty. Once they are awake they return much more rapidly than the healthy child into a state of sleep and coma, after a brief period of pitiful crying. Even in an older child suffering from slight degrees of sluggishness of reaction it might be difficult to determine whether or not toxic conditions exist. If the children are occupied and someone plays with them, a large part of the toxic signs disappear in the first hours of the disease. The children can temporarily appear relatively alert. However, left to themselves they immediately relapse into a state of apathy, with the staring glance or the aimlessly wandering eyes, directed into the distance, demonstrating the lack of interest in the surroundings. Objectively the dimming of consciousness can to a certain extent be determined by the delays with which the child reacts to painful stimuli with defense motions and by the speed with which he relapses into the state of apathy after the cessation of these stimuli.

The onset of apathy is accompanied by a number of other symptoms. Temperature elevations have already been mentioned. However, rises in temperature do not occur regularly in all infants suffering from toxic gastro-enteritis. Only some of them show increases up to 39 to 40°. In other children the temperature reaches about 38 to 39° and finally there are some whose body temperature remains normal or even becomes subnormal. It is therefore not possible to draw conclusions from the course of the temperature curve on the severity of the disease or the prognosis.

Severe disturbances occur in the organs of circulation. The pallor and coolness of the extremities as signs of the failure of the vasomotor centers have already been mentioned. The heart in these children is by percussion and roentgenologically small, probably corresponding to the decrease in the quantities of blood in circulation. The smallness of the heart is in part simulated by a swelling of the lungs which can be recognized by percussion and in the roentgenogram by a lowering of the diaphragm and the clear lung fields. This dilatation of the lungs which is particularly pronounced in the toxic phase after infections caused by bacteria of the shigella groups, is probably the result of a toxic irritation of vegetative centers. The disturbances in the circulation explain in part the pathological symptoms and the disturbances in the metabolism of these patients.

Signs of more or less severe prostration are always present in infants suffering from toxicosis. The children are in a state of collapse. The nose appears pointed; skin color is greyish, the eyes appear haloed. The extremities are cool. The heart beat becomes slower. Signs of failure of the vasomotor centers appear and during the first phase of the disease endanger the life of the children, particularly those whose nutritional state had already been damaged. The patient loses much weight. These weight losses are the results of severe disturbances in the salt and water metabolism of cells and tissues (see later). Weight drops of several hundred grams within a few hours or days are not rare. The amounts of weight decreases are determined in the individual case by the age of the child, his nutritional state and his constitution. The youngest infants, children whose nutritional state is damaged or who are constitutionally hydrolabile suffer greater weight losses than older children in a good nutritional state who had maintained the ability for water absorption in the tissues. As accompanying symptom of the state of shock which finally occurs and as a result of the weight drops a number of further changes appear. The fontanel is depressed considerably. The muscula-

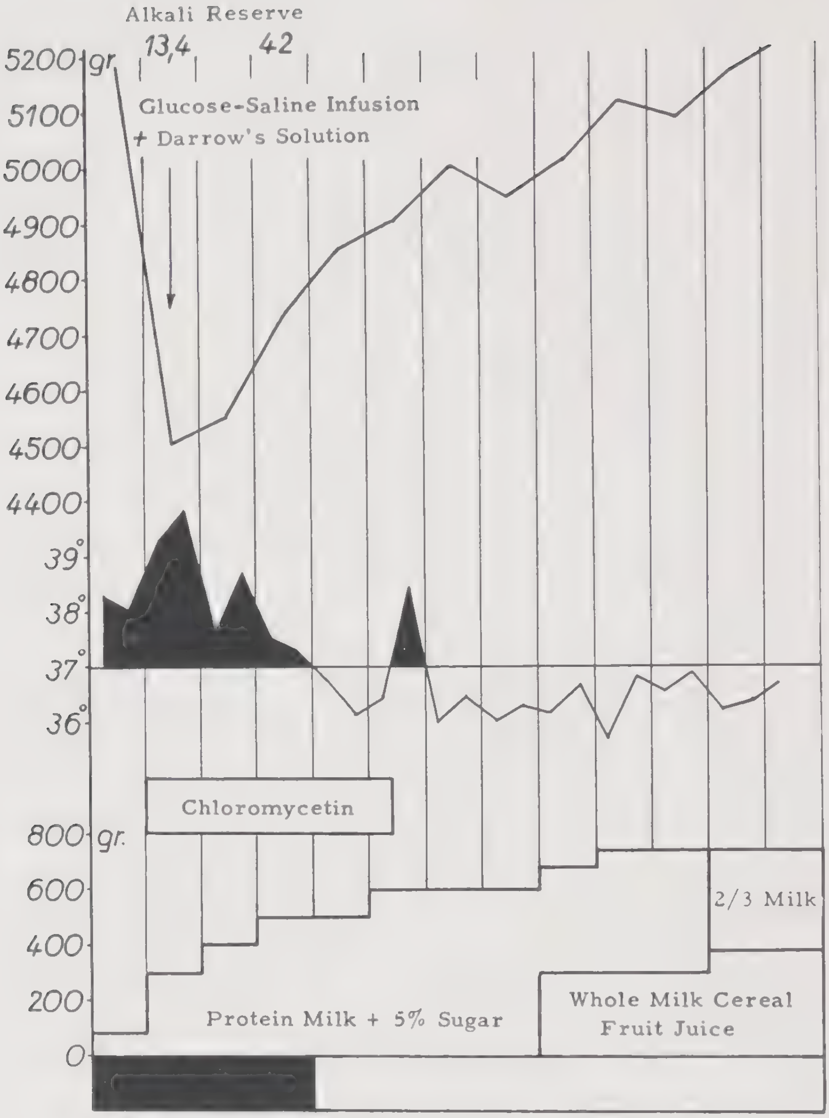

Fig. 40. Infant A.G., seven months old. Toxic gastro-enteritis. Dehydration and acidosis. Cure after starvation period with tea only. Protein milk and 5% sugar. Chloromycetin.

ture becomes rigid, hypertonic. Raised skin folds on abdomen or on the extremities fail to return to normal. As an ominous sign the skin loses its normal elastic consistency. Subcutaneous tissue and skin appear thickened, wax-like and sclerematous. The changes which are observed in the visible and palpable tissues occur probably to a similar degree in the tissues of the internal organs. In part the cloudy swelling of internal organs as observed during the post mortem examination of a child who had died of toxic gastro-enteritis is analogous to the changes observed on skin and musculature in living patients. It can be definitely assumed that all these changes bring about a disturbance in the functioning of the organs and tissues. The weight losses of toxic infants can occasionally be overlooked because of edema formation. The severity of edema in those children who lose only insignificant amounts of body weight is, however, so pronounced that they can easily be observed in form of doughy swellings on the lower part of the legs and the back of feet and hands.

Diarrhea and gastric symptoms are never absent in children with toxic gastro-enteritis. Vomiting can be so severe that no food at all is retained. Bilious vomiting occurs in a few patients already at an early stage. Vomiting of coffee-ground-like material (vomitus of blood) is prognostically an unfavorable symptom. The intensity of diarrhea varies and does not permit any conclusion regarding the severity of the disease. Greenish, mucous, shreddy, watery, and foamy stools, containing very little solid substance, are usually eliminated in great number at the height of the disease. In the microscopic picture of the stool we find few red and white blood cells as the expression of an inflammatory intestinal irritation. The reaction of the stool is usually acid at the beginning of the disease. After the diarrhea persists for a while, we also find eliminations with alkaline reaction, probably as a result of large quantities of alkaline mucus and alkaline intestinal juices which are secreted from the irritated mucous membrane.

Abrupt weight loss and dehydration, collapse and shock, dimming of consciousness, fever, diarrhea and vomiting are the pathological symptoms which permit the diagnosis of a toxic gastro-enteritis or intoxication, even without any further analysis.

In addition to these obvious pathological symptoms which can be readily recognized without difficulty upon examination of the patient, there are a number of symptoms which, however, can be determined only with the aid of special laboratory tests. It is important to recognize the changes resulting from severe disturbances in metabolism since their treatment is urgent and it is not always possible to wait for the results of laboratory examinations before instituting the necessary therapy. Acidosis must be mentioned here in the first place. The diagnosis of acidosis is made objectively by the finding of a decreased alkali reserve in the blood (less than 40 vol.%). The picture of the condition includes symptoms which for the physician make the existence of acidosis likely. Acidosis manifests itself by changes in the type of breathing, which has been described as toxic breathing. Each individual breath is strikingly deep and at times the rhythm of breathing is slowed down. The manner of breathing does not correspond to the normal rapid breathing of the healthy infant. The chest rises considerably and falls again with every breath. If the temperature is very high the rate of respiration is accelerated so that the breathing can then correctly be compared to that of "pursued game." The children breathe with their mouth open. Despite deep and adequate breathing the children are cyanotic or there is a striking contrast between the greyish-blue, pale and marbleized skin and the bright red lips. In severe cases and with unfavorable course of the toxicosis the intervals between the individual respirations become longer. The occurrence of this type of breathing, resembling the Cheyne-Stokes breathing and finally a gasping respiration, is the result of a paralysis of the respiratory center. Prognostically it is an unfavorable sign.

Anoxemia leads to damages of the heart action and to the accumulation of large quantities of blood in the abdominal organs. Meteorism develops. The abdominal walls are taut. In emaciated dystrophic or atrophic children the lively peristalsis of the intestines becomes visible through the thin, fatless abdominal walls so that one might suspect an obstruction in the intestinal tract if the violent diarrhea and the appearance of peristalsis in different and varying places would not speak against this assumption. In a few patients, however, a real paralytic ileus develops sooner or later in the course of the disease which involves not only the small intestines but also the stomach in form of a stomach atony. Percussion reveals a tympanism. The boundaries of the stomach are enlarged.* The children become grey and

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* For an approximate determination of the boundaries of the stomach it is recommended to place the stethoscope below the sternum and then approach the stethoscope with scratching motions from the middle or lower abdomen. Upon reaching the limits of the stomach the resonance changes by suddenly becoming lighter and louder. Comparisons of the limits obtained by auscultation with the roentgenogram confirm the correctness of the results.

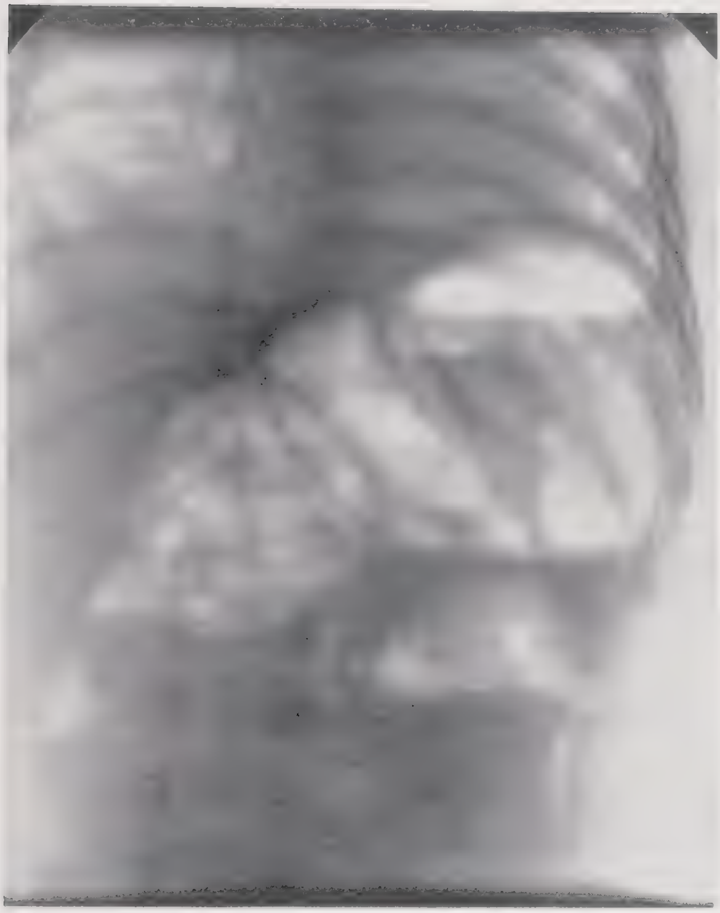

Fig. 41. Infant A.W., four months old. Pseudo-ileus at the onset of an acute gastro-enteritis. Vomiting. One day severe diarrhea, then constipation. Meteorism. Distended, tender abdomen. Visible peristalsis. In roentgenogram multiple fluid levels. Irrigoscopy: normal filling of the large bowels. No passage of barium in lower small intestines. Diagnosis according to roentgenogram: obstruction in small intestines. Surgery because of suspected mechanical ileus. Severely dilated loops of bowels with spastically contracted loops in between. No mechanical ileus. After four days, severe enteritis.

cyanotic. Breathing seems difficult. Great restlessness and fear accompany this serious picture. The abdomen, particularly the upper abdomen, is distended and if a stomach tube is introduced large quantities of greenish-brown liquid are eliminated together with much gas. At this stage infants frequently do not vomit or they eject only small quantities of bilious stomach content together with brownish-red blood. The paralytic ileus can, if it appears at the onset of the disease, simulate the picture of a mechanical ileus so that it is impossible to differentiate it either by the clinical picture or by roentgenogram. The diarrhea ceases and the children become constipated. They vomit and appear greyish and prostrate. The distended intestinal loops filled with liquid, simulate a tumor. Fluid levels are observed in the roentgenogram. The picture of this "pseudo-ileus" can resemble a real ileus to such an extent that a decision to operate becomes necessary in order to clarify the diagnosis. Upon opening of the abdominal cavity small quantities of serous liquid are found. The intestines are strongly injected. A severe dilatation of the intestinal blood vessels exists. Several sections of the small intestines are spastically contracted, others strongly distended, resulting in a picture which could best be compared to sausages which are tied to each other. In severe toxicosis with acidosis these conditions occur not infrequently already at the onset of the disease. More frequently they appear as complications in a somewhat later stage of the disease.

The urine of infants in toxicosis contains, as a result of damages to the capillaries and kidney epithelium, protein, cylinders, leukocytes and erythrocytes in large number. This symptom of kidney irritation disappears only five to eight days after the onset of improvement. The reaction of the urine is mostly acid, occasionally, however, neutral or alkaline.

Glycosuria occurs in the urine at an early stage, not infrequently already in the dyspeptic preparatory phase. The secretion of sugar is not the result of a metabolic disturbance or a disease of the pancreas. The sugar which is eliminated is not glucose but those types of sugar which were ingested with the food; this is a result of damage to the kidney functions.

The following must be observed for testing for glycosuria: lactose and maltose reduce just like glucose, cane sugar does not reduce. It is therefore necessary in the testing for glycosuria by the reductive ability of the urine to split the double sugars by acidification and later neutralization.

The blood of the children reveals regularly a strong leukocytosis and a clear shift to the left of the white blood cells. The protein fractions are changed in the sense that easier precipitated fractions predominate. The routine liver function tests reveal in part of the children pathological values, but it is not possible to draw conclusions from these results as to the severity or the prognosis of the disease.

Although more detailed analyses of the pathological condition will always reveal new changes in the organism of the toxic infant, the searching for further symptoms is of no practical significance in arriving at a diagnosis. The diagnosis of toxic gastro-enteritis or "intoxication" is based on the symptoms of apathy, shock, abrupt weight drops, disturbances in circulation, dehydration, toxic breathing, acidosis (anhydremic anoxemic condition) and possibly fever, accompanied by diarrhea and vomiting.

It is not possible to explain the impressive pathological symptoms of toxic gastro-enteritis with characteristic pathologico-anatomical findings. All examinations in this direction, although carried out with best possible technique, have failed. Not even the intestinal tract of these children reveals any pronounced changes. In addition, it is impossible to differentiate between changes which were already present during life and those secondary changes which developed during agony in the non-resistant tissues. The slightly hyperemic intestinal mucosa reveals exudative processes, a varying content in the mucosa of leukocytes and hemorrhages and hemorrhagic erosions in the uppermost layers of the mucosa. Fine fibrinous membranes may be observed over small areas. The lymphatic apparatus of the intestines is swollen. The peritoneal covering of the intestines shows frequently peach-colored injection as described in real cholera. The mesenteric lymphnodes show enlargement with whitish fatty tissues. The other organs reveal signs of cloudy swelling. The kidneys show fatty degeneration, at least on microscopic examination. Not infrequently they contain microscopically visible centers of infiltration. Despite the albuminuria and cylindruria in the urine, however, there are never changes which could be interpreted pathologico-anatomically in the sense of an acute nephritis. The adrenals, on the other hand, show changes, with the lipoid content considerably reduced, a finding which in view of the many clinical symptoms which point to a disturbance in the vegetative system, is not without interest. The

> liver reveals serious changes. A strong fatty degeneration of the liver might perhaps be the expression of an attempt by the organism, deprived of carbohydrates, to substitute the missing carbohydrates in the liver by rebuilding them from the fat mobilized from fat depots. Whether this fatty degeneration of the liver represents also the expression and result of anoxemia and thus of a serious disturbance of the liver functions, has not been proved.
>
> Only occasionally we find in severely damaged children and in individual epidemics greater anatomical changes without proof of a specific bacillus (shigella infection). Swelling of lymph follicles and plaques in the small intestines are observed. Ulcerative lesions of large parts of the jejunum appear. There is gas formation in the intestinal wall so that there appear pinhead to pea-sized blisters thickly spread over the mucosa.

Examination of the entire metabolism has also not brought any results which might serve to explain the serious pathological processes in alimentary intoxication. In contrast to acute dyspepsia, we find changes in the metabolism which point to a tissue decomposition (see pathogenesis). The absorption of most food components is disturbed. This applies above all to the nitrogen balance and the mineral substances with the exception of calcium and phosphates. The substances which are necessary for the building of protoplasm are lost. And since in part the quantities of potassium, sodium and chlorine which are eliminated are larger than those contained in the ingested food, they must originate from the reserves contained in the cells and tissues. In contrast to the healthy child, the greatest part of the salts is eliminated with the stools while the quantities of salts leaving the body with the urine decrease considerably. The existence of acidosis in children with alimentary intoxication, which can be proved by the increase of the acid reaction of the urine, the high ammonium coefficient and the lowering of the alkali reserves, speaks for the occurrence of alkali losses in the intermediary metabolism. On the other hand there is no ketonuria as exists in other acidotic metabolic disturbances (diabetes). The accelerated peristalsis leads to the appearance of large quantities of unsplit fat and possibly carbohydrates in the stools.

Experiences of earlier years justify the assumption that the essential cause of toxic gastro-enteritis is inadequate nutrition.

The syndrome was therefore described as alimentary intoxication. Today, however, it seems justified to emphasize the significance of specific and non-specific enteral infections as etiological factors in toxic gastro-enteritis. This does not reduce the significance of the alimentary factors. The facts that almost only artificially fed infants suffer from toxicosis, that the incidence of toxicosis decreases with proper milk hygiene, that faulty care of the infant which involves the digestive processes (e.g., overheating) favors the development of toxicosis, show the significance of the alimentary functions in the pathogenesis of the disease. In all these disturbances, however, the previously described settling of the intestinal tract with bacteria plays a decisive role. The syndrome of toxic gastro-enteritis cannot be separated from the influences of specific or non-specific infections. The formerly supported theory that toxicosis is a type of septic process must, however, be rejected, although bacteria can occasionally be found in the blood of seriously ill children; their significance as etiological factors of an acute gastro-enteritis has so far not been proven.

The role of the bacteria in the small intestines is perhaps only that of a doorkeeper who, because of damage to the intestinal wall, opens the door to permit the entrance of physiological and pathological waste products of food protein into the circulation.

The first result of the damaging influence upon the tissues by heterogenous substances, whether they are bacterial toxins, endotoxins or protein waste products from the food, is a disturbance in the water balance as has already been mentioned. The great strain on water metabolism is the cause for disturbances which in infants appear earlier and for lesser causes. Diarrhea and a pathologically increased insensible perspiration lead to considerable losses of water, particularly if water binding salts (Na, K, bicarbonate) are lost simultaneously. Water losses due to violent and repeated vomiting are accompanied by losses of salts resulting in a metabolic alkalosis since in place of Cl, which is lost with the vomitus, HCO_3 appears in the blood. Water losses caused by diarrhea and a pathologically increased insensible perspiration with its consequences for circulatory and kidney functions, result in dehydration and finally in acidosis. Whether the shift in metabolism takes place in the direction of acidosis or of alkalosis is determined by the extent to which vomiting on the one hand. or diarrhea and disturbances in insensible perspiration on the other hand dominate the pathological picture.

The shifting of the water is further accompanied by an exchange of salts from one to the other of the three fluid reservoirs (circulation, intercellular fluid and intracellular fluid). Potassium travels from the cells into the intercellular space and into the plasma. In the cells potassium is replaced in part by sodium. The potassium which migrated from the cells is eliminated by the kidneys. In this manner a potassium deficiency develops not only in the cells but finally also in the peripheral and circulating fluids. Hypokalemia in the plasma causes severe disturbances if the potassium losses are not replaced. The reduction of the potassium content of the plasma is of more importance for the appearance of pathological developments than the potassium losses from the large potassium reserves which the organism maintains in the cells. Potassium losses in the periphery lead to atonic conditions of the musculature. The atony of the striated musculature can reach such a degree that it resembles flaccid paralyses. The heart muscles also suffer. The reduction of the potassium content of the serum but not of the muscles leads to changes in the electrocardiogram, which shows a lowering of the S-T segments and a small, completely absent or even reversed T wave. Intake of potassium immediately removes the changes in the electrocardiogram. Systolic murmurs can be heard and the heart is dilated. The blood pressure comes down, and gallop rhythm and finally cardiac arrest may result. In the intestinal tract the tonus of the musculature of stomach and intestines is lowered. The peristalsis is changed. A paralytic ileus develops. Pathological symptoms resulting from potassium deficiency occur when the content in the serum is reduced below the normal values of 8 to 10mg% (= 2.05 to 2.56 milliequivalent per liter).

Transition from dehydration to acidosis takes place when the easily available fluid quantities in blood circulation and intercellular space are no longer sufficient to balance the large water losses in order to maintain the osmotic pressure in the periphery. The result is thickening of the blood, hemoconcentration. The circulating blood volume decreases. The distribution of the blood suffers. Large quantities of blood accumulate in the mesenteric vessels. The children now appear pale, greyish and livid. Anoxemia develops. The insufficient oxygen supply, together with the dehydration, affects the kidney function. The urine quantities are reduced to the point of anuria. The small quantities of concentrated urine contain cylinders, erythrocytes, protein and sugar. The carbon dioxide elimination through the lungs is reduced. The result is acidosis.

The accumulation of acid metabolic products and the anoxemia represent a severe damage to the vegetative nervous system and to the centers which direct the circulation. In the tissues which are inadequately supplied with oxygen the capillaries become permeable. Similar to any other state of shock this phase of the disease, which has also been described as serous inflammation, is accompanied by loss of fluids from the tissues. Large quantities of water change location. The fluid content in the cell is reduced. In the intercellular spaces the water is no longer retained. In the peripheral circulation, too, water is lost which can no longer be replaced. If the process has deteriorated from dehydration to acidosis and further to toxicosis, a vicious circle develops whereby the acidosis is increased due to further and more severe damages of the cells. This in turn aggravates the state of shock and brings about a continuation of the toxic condition.

Any feeding mixture can bring about an intoxication if it reaches the intestinal tract at an unfavorable time. Not the foreign, heterogenous nature of the food or the fact that it is spoiled is decisive. An increase in food intake or the presence of food substances in the intestines are only secondary causes for the development of intoxication if the intestines are settled with bacteria and damaged. On the other hand—and this seems to further emphasize the significance of food—discontinuation of food intake leads to the disappearance of symptoms of intoxication. Discontinuation of food intake is one of the prerequisites for curing intoxication. The development of an intoxication becomes most apparent when intoxication occurs as a result of a quantitative increase of a certain food substance which had already caused an acute diarrhea in smaller quantities.

Any errors in quantity and quality of feeding mixture promote the settling of the intestines with specific and—particularly in young infants—non-specific bacteria. The settling of duodenum and small intestines with bacteria remains in every case the first factor which induces a chain of processes which culminates in the picture of a toxic condition. Schematically the processes can be presented as follows:

Bacterial invasion of the small intestines
↓
Absorption of bacterial toxins
(or protein waste products)
Liver damage
↓
Freeing and egression of water from interstitial
tissues and cells
Electrolyte loss (Potassium) and migration
↓
DEHYDRATION
↓
Thickening of the blood. Reduction of
circulating blood volume—Anoxemia
↓
ACIDOSIS
↓
Damage to nervous centers, kidneys and heart
↓
Shock
↓
TOXICOSIS

The symptoms of intoxication could be compared to coma due to acidosis. The metabolic acidosis in the organism, which can be proved, justifies this comparison to a certain extent. There are, however, no analogies to a primary metabolic acidosis as exists for instance in diabetes. Hunger, which aggravates the acidosis of the diabetic patient, cures the state of intoxication. There are no acetone bodies in the urine of a child suffering from intoxication.

Toxic gastro-enteritis can progress to any of the three stages of dehydration, acidosis and toxicosis. There are patients whose condition does not deteriorate beyond the stage of dehydration. In these children it is relatively easy to remove the pathological condition within a few hours by adequate fluid intake (together with discontinuation of food intake). If a state of acidosis or even toxicosis has been reached, other measures are necessary in order to repair the metabolic disturbance and the severe damages to the tissues. The fate of a child therefore depends essentially on how early the physician or those taking care of the child recognize the impending catastrophy. In a child who is in a good nutritional state it is then still possible to prevent the development of a severe state of intoxication. During the period of lability of the intestinal processes (diarrhea, pallor, lack of appetite, vomiting, restlessness) any change in diet should be carefully considered. During this time increases or essential changes in the food should be particularly avoided. In this manner the toxic gastro-enteritis can be evaded, at least in eutrophic and the majority of dystrophic infants. Prevention of intoxication caused by infection is not possible with similar certainty.

Treatment of alimentary intoxication has to fulfill two tasks: 1) to re-establish the sterility of the intestinal tract, and 2) to remove the state of dehydration and acidosis which developed as a result of a settling of the intestines with some species of germs. The fulfillment of both tasks has today a more certain chance of success than even a few years ago.

The fight against dehydration and acidosis is the first aid in the treatment of any acute toxic gastro-enteritis. "Sterilization" of the intestines which is also part of any initial treatment of toxic gastro-enteritis requires a certain amount of time before the effects of chemotherapeutics or antibiotics succeed in re-establishing the bacterial balance in the intestines.

Restitution of a low bacterial count in the small intestines can be accomplished in two manners:

1) By an interruption in food intake, during which period only tea is given: this means the discontinuation of any food which might serve as nutrition for the bacteria in the duodenum and small intestines. This hunger period during which the infant

receives only tea sweetened with saccharine for eight, 12 to 24 hours, depending on age, nutritional state and severity of disease, was formerly the only method of expelling the bacteria from the upper intestinal sections. Giving water or tea sweetened with sugar, which has been recommended by some authors, does not completely fulfill this requirement. The use of a highly concentrated (20%) sugar solution is permitted only in older children. In the case of all young and severely ill children it is not advisable to choose any but a radical method. The dangers of absolute hunger (with the exception of adequate water intake) have disappeared today since it is now possible to supply the organism by parenteral infusion, together with the water supply, with salts and sugar as well as assimilable protein in adequate quantities.

2) The use of sulfa preparations and antibiotics has today essentially facilitated the restoration of sterility of the intestines. The use of the individual sulfa preparations and the different antibiotics has been described earlier (see p. 367). Depending on the severity of the toxic gastro-enteritis the physician will prescribe only sulfa preparations or will use antibiotics, alone or in addition to the sulfonamide drugs.

The above data can be summarized in the following table:

	Light Cases	*Severe Cases*
Young infants	Sulfathiazole, Sulfadiazine (0.2g per kg. body wt.) Streptomycin per os 4 × daily 50 mg	Sulfathiazole or Succinylsulfathiazole (0.3g per kg. body wt.) Streptomycin or Chloromycetin (50mg per kg. body wt.)
Older infants	Sulfathiazole or Sulfadiazine	as above

Therapy with the above preparations should be continued until the stools are "normal" for at least two days, that is, that only one, two or three pasty stools are eliminated. If one of the preparations used does not bring about an improvement in the stools after four to five days, the drug should be replaced by one of the other preparations since the preparation used was not effective in that individual case for fighting the intestinal infection, or the intestinal bacteria have developed a resistance against the drug.

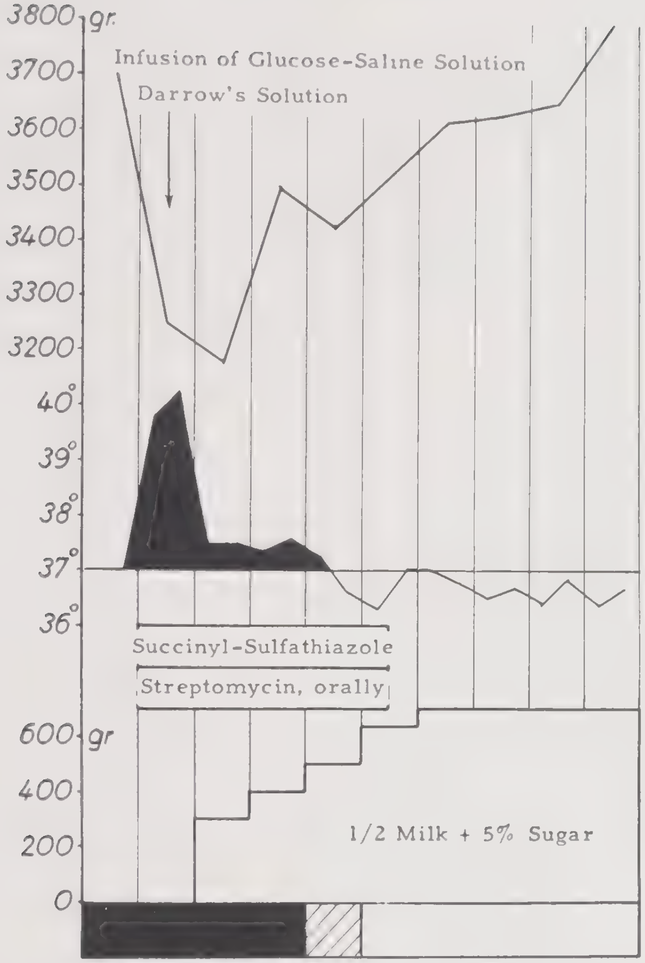

Fig. 42. Infant G.S., five weeks old. Eutrophic up to the time of present illness. Toxic gastro-enteritis. Acidosis. Convulsions (spinal fluid: Pandy +, $^2/_3$ cells). Treatment: starvation period of eight hours with tea only. Infusion of glucose-saline and Darrow's solution. Succinylsulfathiazole 4 x 0.25g. and streptomycin per os 4 x 50 mg. $^1/_2$ milk plus 5% sugar in rapidly increasing quantities.

3) Simultaneously and of at least the same importance stands the need for restoring the disturbed water and salt metabolism. The parenteral supply of fluids, salts, nutritive substances and of vitamins is the most urgent task in the beginning of the treatment of a toxic gastro-enteritis.

There are several methods of supplying fluids parenterally:

a) The subcutaneous infusion of a physiological salt solution or Ringer's solution has at present been abandoned. The infusion is painful and permits only the supply of relatively small amounts of fluid. The large accumulation of fluids in the subcutaneous tissues which causes a tumor-like swelling of the skin produces a considerable chilling, particularly if the absorption of fluids in the damaged circulation takes place only very slowly.

Today the obsolete method of subcutaneous infusion returns in a somewhat altered form. An enzyme was found in form of hyaluronidase which, if added to the injected fluid, accelerates the absorption considerably. By using this enzyme it is possible to effect complete absorption of 150 to 300cc of fluids within 10 to 20 minutes. For patients with particularly large and dangerous fluid losses and dehydration the subcutaneous infusion with the addition of hyaluronidase can today be recommended.

The technical execution of subcutaneous infusion with the addition of hyaluronidase is simple. From a sterile container the fluid runs through a y-glass piece to two rubber tubes with large needles. The needles are introduced subcutaneously, preferably in the abdominal wall to the right and left of the umbilicus, and attached with tape. At the beginning of the infusion $^{1}/_{2}$ mg hyaluronidase dissolved in 1 cc distilled water is rapidly added through each of the rubber tubes into the running stream of fluid which is to be injected. The absorption of the fluid is then surprisingly rapid, without the development of any visible changes in the skin which after the infusion appears only slightly reddened and warm. Salt-glucose solutions or plasma can be used for the injection.

b) The intravenous infusion after introduction of a suitable needle into one of the veins of the skin of the skull or any other part of the body or into a vein following a venesection is today the most widely used method for rapid and prolonged infusion of fluids. The liquid flows by drops from a burette into the vein or it can be rapidly injected with a syringe. The advantage of this method is that the injection can be extended over many hours and even days. Inflammation and thrombosis of the vein occasionally occur after a longer period of time. They can, however, be cured rapidly with wet dressings and the use of antibiotics (penicillin). The disadvantage of the method is the fact that a small surgical apparatus must be prepared for the venesection and that particularly in the smallest infants with a bad circulation the preparation of an intravenous infusion requires considerable experience. In infusions by venesection the necessary, prolonged fixation of the child is frequently troublesome and difficult.

c) A third method is the infusion into the bone marrow of one of the long bones.

Technically the puncture of the bone marrow of tibia or femur or, in case these bones had already been used for infusions, puncture of the marrow of the iliac crest is the simplest in the infant. The puncture of the tibia takes place approximately on the border between upper and middle third, exactly in the center of the planum tibiae. Infusion in the femur takes place preferably from the outside of the femur in the upper section of the lower third, and not from the front, in order to avoid injury of the bursa patellaris which occasionally extends considerably upwards. For the puncture of the leg, which is fixed on a splint, not too thin a sternal puncture needle is used with mandrin. The needle is introduced under careful rotating pressure through the corticalis, in young infants without and in older infants with local anesthesia. Upon reaching the marrow a noticeable decrease of resistance, frequently connected with a crackling sound, is encountered. The mandrin is now removed. By aspirating marrow with a syringe, half filled with the liquid which is to be infused, one ascertains whether the needle is located in the marrow. At times it is not possible to aspirate the marrow. It is then sufficient to try to inject the fluids, after the air from the needle has been removed by dropping a small quantity of a saline or similar solution on the hub. If the injection succeeds without resistance the needle is quickly connected with the connecting tube of the burette which is filled with the injection fluid. The air has to be removed beforehand from the entire system. The hydrostatic pressure of the liquid should amount to about 100 to 120 cm. Needle and connecting tube of the burette are connected with strips of adhesive tape. The area of the puncture is protected from infection by sterile gauze. The speed of infusion is then regulated on a drop counting device, depending on the condition of the child and the type of infusion fluid used. In order to avoid

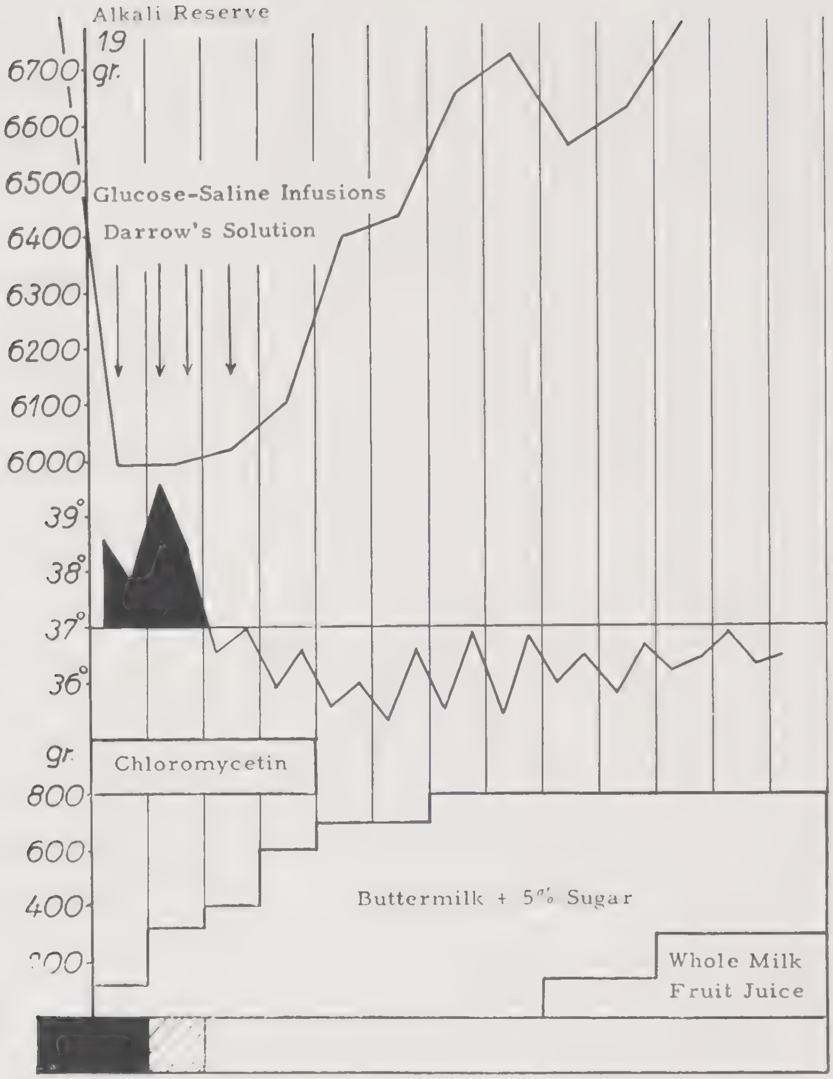

Fig. 43. Infant M.S., six months old. Toxic gastro-enteritis. Dehydration of long duration. Acidosis (alkali reserve 19). Cure by repeated infusions of glucose-saline solution; at first twice daily followed by infusion of Darrow's solution. Starvation period with tea only. Increasing quantities of sweetened buttermilk. Chloromycetin.

infection on the point of puncture the needle must not remain in place for more than 12 to 18 hours. In case a continuation of the infusion is necessary beyond this time the infusion should be continued on another bone (not on a different place on the same bone). Contra-indications for the application of intratibial infusion are all septic processes which also includes acute infections of the salmonella group (typhoid, paratyphoid). In these patients a periostitis or osteomyelitis occasionally develops at the area of injection. The infusion of liquid into the marrow with a syringe causes severe pain due to the sudden increase in pressure in the marrow cavity. The infusion of blood into the marrow is not recommended since the erythrocytes which settle as sediment in the burette and the connecting tube frequently obstruct the system and make the continuation of the infusion impossible.

The choosing of the fluid used for parenteral fluid supply is not done arbitrarily. It must be chosen depending on the condition of the patient and the type of metabolic disturbance.

The first task is always the treatment of the dehydration. This accomplishes primarily a replenishing of the circulation and the intercellular fluid reserves. For this purpose isotonic salt-glucose solutions, e.g., Glucose 41.0g, Phys.NaCl solution 200.0 ml, Aq.dest.ad 1000.0 ml, and human plasma, which should if possible be of the same group as that of the recipient, have been found suitable. Once the dehydration has been removed the second task in the infusion therapy is an attempt to treat the acidosis and to re-establish a normal electrolyte balance in the cells. For this purpose solutions as recommended by *Hartmann* and *Darrow* have been found useful.

Hartmann's solution is intended to fight the acidosis. It contains per 100 cc:

Sodium lactate	0.31 g
Sodium chloride	0.60 g
Potassium chloride	0.03 g
Calcium chloride	0.02 g

Darrow showed that the cell membrane becomes permeable for potassium, that potassium is lost in large quantities and that these losses are reversible. On this basis it is recommended to add potassium to the infusion solution. This solution contains: 40 cc molar Na lactate plus 2g KCl plus 3g NaCl plus 710 cc water or 4.4g sod. bicarbonate (= 52 m Eq/1), 2.7g KCl (= 36 m Eq/1) plus 3g NaCl (= 69 m Eq/1). For diluting the salt mixtures a 5% glucose solution is recommended which should facilitate and accelerate the transition of potassium from the serum into the cell. *Darrow*'s solution improves the acidosis and provides replacement for the extra- and intracellular potassium losses. Infusion of potassium should take place only when the hypokalemia is confirmed in the serum. If this determination is not available or if the results cannot be obtained quickly enough, the infusion can be given on the basis of the changes which can be observed in the sick child such as atony of the musculature, breathing with open mouth resembling a fish mouth, and the changes in the electrocardiogram. In order to avoid potassium poisoning the infusion of a potassium solution can be started only after the kidney secretion has started functioning again.

Compensation of potassium losses can also be effected by giving potassium citricum per mouth, although this method is less certain, particularly if there is severe diarrhea. Four to six times daily the children receive 0.25–0.5g potassium citricum until the symptoms of potassium deficiency are improved.

If it has been possible in this manner to re-establish the water and electrolyte metabolism and the normal reaction in all three fluid reservoirs (circulation, intercellular and intracellular fluid spaces) parenteral feeding with amino-acid hydrolysates (e.g., Amigen) is resumed in those patients who because of severe gastric symptoms cannot yet be fed per month. In this manner it is possible to maintain many infants, who at times cannot be fed per mouth, alive for many days without essential weight losses.

It does not always appear advisable to continue the infusions for many hours or even days. At the time when the functions of water and salt metabolism are not yet restored prolonged infusions cause the losses of mineral substances (sodium, potassium, etc.) through stools and urine. Hydremia, hyposalinemia and edematous conditions develop. If the fluids are supplied in excessive quantities and too rapidly, dangerous secondary symptoms such as pulmonary edema and disturbances in the circulation can develop. Hypoproteinemia following prolonged infusion can reach unpleasant degrees. A control of serum protein is always necessary in prolonged continuous infusions. It seems advisable to introduce pauses between the infusions after the acute state of dehydration has been overcome.

The amount of fluids to be injected depends on the condition and the weight of the child. The quantities should not be too small. Overcoming of dehydration manifests itself in the disappearance of the dryness of the skin. Raised skin folds straighten out rapidly again. The fontanel is no longer sunken. The eyes are no longer haloed. The pulse becomes stronger, bradycardia disappears. The cardiac dullness or, presented more objectively, the dimensions of the heart in the roentgenogram return to normal size. The children again appear rosy and the extremities feel warm. Signs of shock disappear. For removing the disturbances of heart and circulation it is recommended to add Cardiazol to the infusion; in order to replace the vitamin losses and to improve the water absorption Vitamin C (50 to 100 mg) should be added. To mention specific figures: in young infants 150 to 250 cc of salt-glucose solution, in older infants 300 to 500cc of the solutions are necessary in order to remove an average dehydration. These quantities should be supplied in the course of two to four hours in form of intravenous or intraosseal infusion. In the treatment of dangerous acute conditions of dehydration and particularly in case of subcutaneous infusion with the addition of hyaluronidase, the infusion is carried out within a much shorter period of time. Quantities of 150 to 300cc can be infused without difficulties within a short time (half hour).

Solutions which are intended to remove the acidosis and simultaneously replace the potassium losses must be infused much more slowly in order to avoid damages to the child. It is advisable to have acidosis or hypokalemia confirmed by laboratory examinations. It is, however, possible in almost all cases to recognize these changes in the blood chemistry also upon clinical examination of the patient. The correction of the acidosis manifests itself in the cessation of deep breathing. The children breathe at a normal rate, more superficial and with closed mouth. The grey, livid coloring disappears. Hypotonia caused by the potassium losses shows improvement. Atonic conditions in the gastro-intestinal tract are replaced by a normal tonus of the intestinal wall. Meteorism disappears. The vomiting of brownish, bilious substances ceases. Bowel movements which might have stopped for a number of hours occur again.

Hartmann's solution, which acts only against the acidosis, or *Darrow's* solution, which is used against acidosis and potassium deficiency, is given in quantities of 60 to 100 cc per kilogram of body weight, depending on the age and the severity of the acidosis; that is, 200–400–600 cc are given very slowly, about 10 to 20 drops per minute, in order to avoid the dangers of potassium poisoning.

	Dehydration	*Dehydration + Acidosis*	*Dehydration + Acidosis + Hypokalemia*	*Hypoproteinemia*
Glucose–Salt	+1	+1	+1	—
Hartmann's Sol.	—	+2	—	—
Darrow's Sol.	—	—	+2	—
Amino-acid Solutions	+2	+3	+3	+3

Explanation: + = Solution indicated; — = Solution not indicated; 1, 2 and 3 = Sequence in which the solutions should be given.

The hunger period should be continued as long as vomiting and lack of appetite exist in the child. If feeding is resumed as long as gastric symptoms exist, two dangerous factors must be considered: 1) the danger of an aggravation or a recurrence of the state of intoxication, and 2) the danger of a stomach atony (see p. 382). Considerable amounts of NaCl are then lost with the large quantities of gastric juices, and hypochloremia and occasionally alkalosis can develop. During the first days of the disease it might become necessary to repeat parenteral fluid therapy if the signs of dehydration and acidosis have not completely disappeared or if they recur.

If there are no signs of a stomach atony and if dehydration and acidosis have been removed following well chosen parenteral supply of water and salts and if the child again eliminates urine which is not of high specific gravity, the feeding of the child may be resumed. The chosen diet which is suitable for treating toxic gastro-enteritis must fulfill two requirements: 1) it must restore and maintain the water absorption in the tissues; and 2) it must reduce the number of stool eliminations. These requirements are not met by breast milk which is poor in salts and protein. Sweetened buttermilk is most suitable. The children receive at first buttermilk plus 5% sugar mixtures in six to eight meals of 20 to 30g each per day. The number of meals depends on the appetite of the child, the size of the meals on the age of the child and the severity of the disease. If the child is very young, the disease severe and the nutritional state reduced to a considerable extent, smaller and more frequent meals are indicated. In children beyond the third month of life the addition of vitamin B and C is necessary, which can best be given in the infusion fluids (50 mg vitamin C) or in form of tablets.

Some authors have recommended to start the nutrition with a carrot soup or a purée of carob flour in place of the hunger period, during which only tea is given, and in place of sweetened buttermilk in small quantities. Due to its abundant content of salt and potassium, pectin or lignine respectively, this food is suitable to exert a favorable influence on the water absorption in the tissues and on the improvement of the stools.

> The best method of preparing carrot soup is in a pressure cooker. For the treatment of toxicosis with carob flour (St. John's bread, Karobon—commercially available under the trade names of Keraton or Arobon) *Glanzmann* has proposed the following routine:
>
> On the first and second day the patient receives no food per mouth. On the second to third day ten times 10 to 20 cc weak tea or half-Ringer's solution is given. Third day: 3% carrot soup plus 3% Arobon, 10 × 40 cc. Then slow transition to skimmed buttermilk (fat content 0.6%), which by progressive concentration is gradually increased to 10%. Aside from this the child continues to receive constant infusions for three to seven days. After this the skimmed buttermilk is gradually, meal after meal, replaced by normal buttermilk (milk 10% plus 3% sugar mixture).

The use of breast milk is not indicated in the treatment of acute toxic gastro-enteritis since in its composition it has neither an anti-diarrheic effect (it contains relatively large quantities of fat and sugar) nor does it promote water absorption (it contains little salt). The use of skimmed breast milk is also not recommended as first therapeutic food in children with toxic gastroenteritis since centrifuged breast milk does not sufficiently improve the water absorption. In very young or in atrophic infants it is, however, advisable to add skimmed breast milk to the buttermilk after the third day of treatment. Breast milk can then contribute its particular favorable characteristics for the vital cell processes.

But even buttermilk plus 5% sugar must be given only in small quantities during the first days of the disease. Any transgression renewed aggravation. In the first 24 hours following the hunger renewed aggravation. In the first 24 hours following the hunger period not more than 150 to 250g, distributed among six to 10 meals, are given. When supplying such small quantities of food it is necessary to cover the fluid requirements of the thirsting organism. Here, too, 150g of fluid per kilogram of body weight must be supplied per mouth or in form of infusions. The further gauging and increasing in food quantities in toxic gastro-enteritis must not take place according to a strict routine, in contrast to the method applied in acute diarrhea. Approximately the following procedure can be recommended:

Total Quantities per Day

First day of food intake:	150–200g buttermilk (with 5% sugar)
Second day of food intake:	150–250g buttermilk (with 5% sugar)
Third day of food intake:	250–300g buttermilk (with 5% sugar)
Fourth day of food intake:	250–350g buttermilk (with 5% sugar)
Fifth day of food intake:	350–450g buttermilk (with 5% sugar)
Sixth day of food intake:	450–600g buttermilk (with 5% sugar)

With this routine the maintenance requirements of the child are met on about the third day of treatment. At the same time the general pathological symptoms and the gastro-intestinal symptoms will have shown an improvement. This permits a greater freedom in the continuation of the treatment.

Individual attention is, however, necessary. The physician can only supervise the careful observation of the patient. Particular care must be taken to search for a recurrence of toxic symptoms. The appearance of the stools must, however, not be decisive in the physician's orders with regard to dietetic treatment. Diarrhea may frequently persist in undiminished force while the entire appearance of the child shows improvement. In such a case an increase in food quantities is permitted. The course of the weight curve shows whether the therapy which had been applied proved suitable. With a turn to the better the abrupt weight loss stops immediately despite initial starvation and despite the very small food quantities given in the beginning. From day to day the weight losses decrease. The weight curve becomes almost horizontal or not infrequently we observe even at this stage

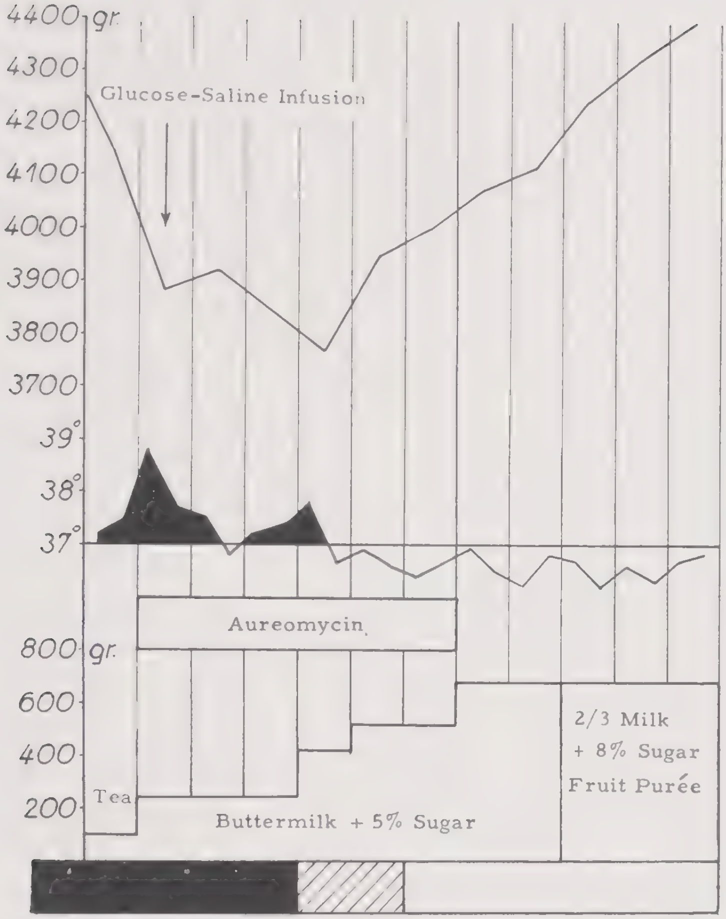

Fig. 44. Infant J.N., two months old. Acute toxic gastro-enteritis. Convulsions. Spinal fluid: Pandy +, protein 50 mg%, NaCl 528 mg%, sugar 66 mg%. Treatment: starvation period with only tea, buttermilk plus sugar in increasing quantities. Infusion. Aureomycin (30 mg per kg of body weight per day).

a weight increase after the interruption in food intake, particularly as a result of parenteral fluid supply. This seemingly paradoxical behaviour—weight increase with calorically inadequate nutrition—is a sign that the cells have regained their ability for water absorption. The stoppage in weight losses may, however, be regarded as favorable criterion in the treatment only if the toxic signs disappear simultaneously from the picture of the disease. The storing of fluids in form of edema can simulate a stoppage in weight loss. The return of the temperature to normal, the better

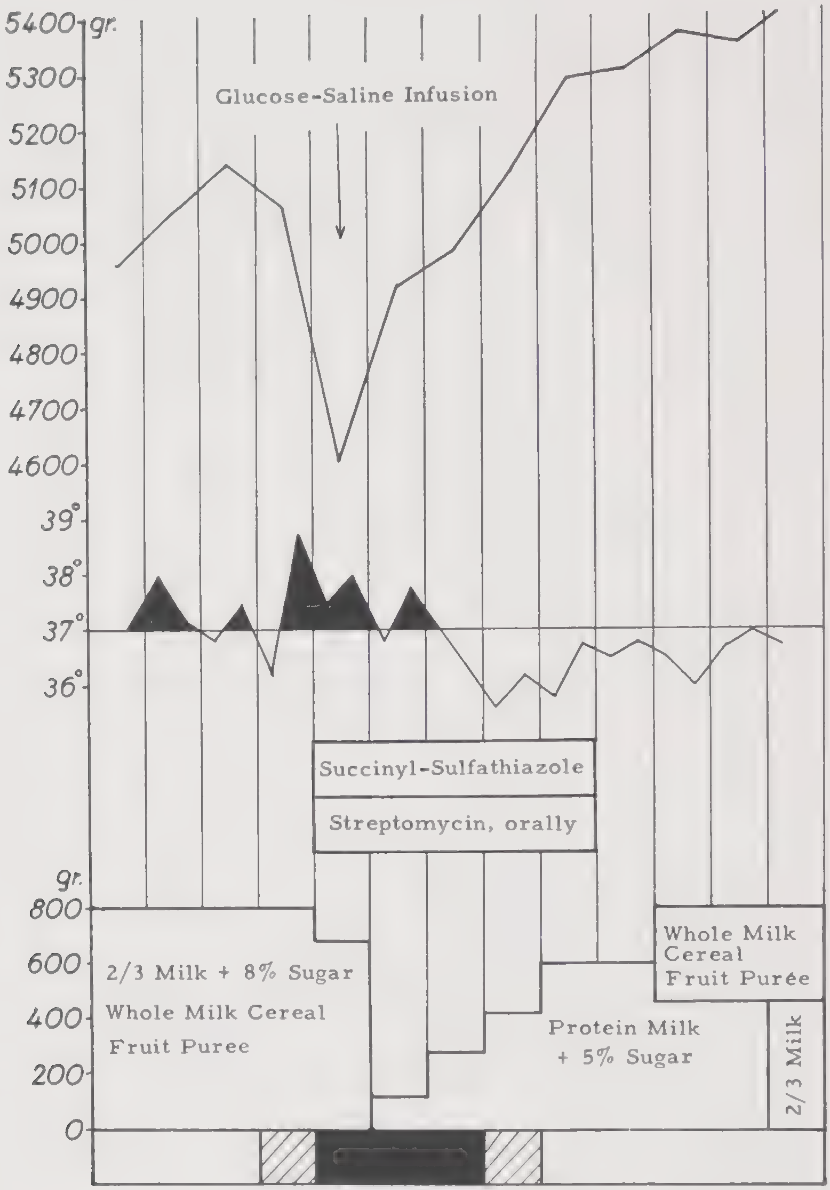

Fig. 45. Infant A.M., four and one-half months old. Gastroenteritis and dehydration. Rapid improvement after infusion of glucose-saline solution. Starvation period with only tea. Protein milk with 5% sugar in increasing quantities.

blood circulation and increased moisture of the skin, the regained interest in the surroundings, the return of a normal type of breathing are further objective signs which permit the conclusion that the intoxication is disappearing.

After the intoxication and the dehydration have been taken care of, the second stage of the treatment, the real restoration, begins. Now the physician has more freedom of action with regard to quantity as well as choice of the type of food. The following possibilities exist for complementing the diet until the quantitative and qualitative requirements are fully met:

1) Transition to breast milk.
2) Transition to protein milk.
3) Enriching the buttermilk with butter or roasted flour-butter mixture.

If breast milk is available it is a valuable complementation of buttermilk, particularly in very young infants and in severely dystrophic or atrophic children in the convalescing stage following intoxication. From day to day 50 to 100g of sweetened buttermilk are replaced by breast milk. A slight initial weight decrease occurs in the first days. This lowering of the weight curve is, however, without significance. The fat, which is now again included in the food, requires a shifting in the salt-water metabolism since the fat needs salts in the intestines which had previously been used for water retention in the organism. After two to three days the weight curve begins to rise again.

If breast milk is not available the sweetened buttermilk which was originally selected as therapeutic food must be supplemented and replaced, about eight to 10 days after beginning of treatment, by the addition of fat (butter) or the addition of sweetened protein milk. Butter is added either in quantities of 1g per 100g of the feeding mixture or better in form of a roasted flour-butter mixture (at first 2g butter, 2g white flour) per 100g of food given with two meals. This supplementation of the diet with fat is gradually extended to all the meals. Replacing the buttermilk with sweetened protein milk is indicated even in cases where the tendency to diarrhea persists. In one meal after the other the buttermilk is replaced by similar quantities of protein milk with sugar. If the stools are already rarer and more solid during the period in which buttermilk is given, the entire food quantity can be replaced by sweetened protein milk from one day to the other. If the weight curve rises and if the stools have become normal, the sweetened protein milk or the buttermilk with fat and carbo-

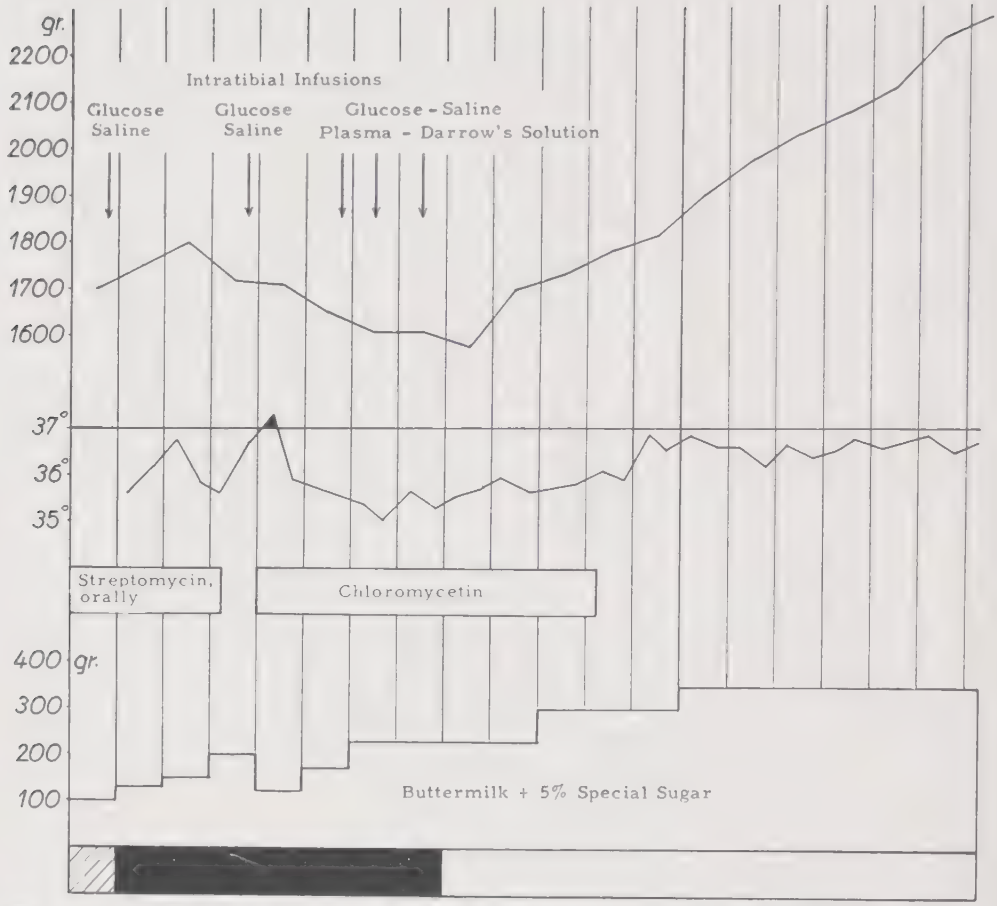

Fig. 46. Infant E.S., one month old premature infant. Birth weight 1700 g. Acute diarrhea changing to toxic gastro-enteritis with dehydration and acidosis. Diet: buttermilk plus 5% special sugar mixture. Treatment with chloromycetin (50 mg per kg of body weight per day) and infusions.

hydrates can be replaced after about 14 to 21 days, depending on the age of the child, by $^2/_3$ milk or by whole milk.

In the infant beyond the third month of life the diet, which in the beginning is qualitatively incomplete, must be supplemented with vitamin C, at first in tablet form, somewhat later in form of fruit juices; in the somewhat older child vitamin B must also be added in form of one of the vitamin preparations, later by egg yolk or bananas. A long period of time is required for complete recovery. The length of time varies depending on the nutritional state of the child at the onset of the acute diarrheal disease. During the period of convalescence, which lasts for weeks, the children still show a strong sensitivity of the intestines. The transition to a normal diet which is suitable for the age of the child can be accomplished only after a number of weeks. In the eutrophic child the therapeutic diet should be continued for about three to four weeks, in the dystrophic child four, six and even eight weeks. Only after this time it is permitted to re-introduce vegetables, larger quantities of fat, etc. in the menu of the child.

The course of a toxic gastric-enteritis is accompanied by a number of complications; their recognition and rapid treatment is of essential significance.

In the first stage of the disease, at the time of dehydration and toxic cell damages, the picture of toxic gastro-enteritis includes the condition of shock. Apart from replenishing the circulation with fluids (see p. 394) analeptica should therefore not be lacking. For this it is more important to provide drugs which support the peripheral circulation than those which affect the heart muscle itself. Cardiazol, coramin, adrenalin etc. are suitable, caffein and camphor are superfluous.

Infants receive 0.2 to 0.3 cc of an 1:1000 adrenalin solution every two to four hours. Cardiazol, coramin etc. is given every three to four hours or more frequently, depending on age, in amounts of 0.2 to 0.5 cc. In prescribing cardiazol and similar preparations one must consider the convulsive action of the drugs which must be counteracted by early and adequate supply of injectable luminal (Phenylbarbiturate, Sevenal). Heat supply by correct nursing care of the children in form of hot water bottles or protracted warm baths are important auxiliary measures in the treatment of shock.

Conditions of shock can recur in young children even during the first days of convalescence, as long as the food intake is calorically inadequate or in case secondary infections occur in the patient. Cool extremities, a cold nose, cold expiration air, cyanosis and occasionally a slowing down of the pulse are signs of threatening collapse of the circulation. The failing of the functions of heart and blood vessels results also in atelectases and so-called hypostatic pneumonias which are confirmed by the dullness or frequently only by fine or medium râles over the lower parts of the lungs. These secondarily infected atelectases are present in all post mortem examinations of children who had died of toxic gastro-enteritis.

Fever, hyperpyretic temperatures and convulsions occur frequently in the first stage of toxic gastro-enteritis. These accompanying symptoms show the participation of the central nervous system in the pathological process. The purely alimentary genesis of fever has at present become doubtful (see p. 345). These hyperpyretic temperatures are influenced neither by interruption in food intake nor by antibiotics or pyramidon. Hyperthermias occur particularly in those patients where the cause of the toxic gastro-enteritis is a specific infection, primarily from bacteria of the salmonella group (see chapter on paratyphoid). Due to central nervous influences complications on the part of the gastrointestinal tract develop at times at the onset, more frequently only after several days. We might mention here once more the atonic conditions which might be aggravated until they resemble the picture of paralytic ileus.

If the intestinal atony extends over the entire intestines and if the peristaltic sounds can no longer be heard the introduction of a stomach tube brings relief or causes the serious condition to disappear. At times the tube must be introduced very deep because of the dilatation of the stomach, before air comes out followed by large quantities of brownish-bloody or bilious liquids. The distension of the upper abdomen disappears. Breathing and appearance of the patient improve. If the atony has progressed to the intestinal sections and if stool retention has occurred the medication of physostigmine (0.1 to 0.2 mg repeated, if necessary, after two to three hours), removes the paralytic ileus. The stomach tube which is preferably introduced through the nose and attached with adhesive tape must remain there for at least several hours until the liquid which is introduced through the tube flows back clear. As long as the tube remains in place and the atony persists food intake is impossible. The atony can last for hours, occasionally for as long as two to three days. During this time the nutritional requirements must be met by parenteral supply of fluids and protein (amino-acid hydrolysate) as well as of vitamins. After the atony disappears small and frequent meals can be given which can be increased only when the symptoms of atony (vomiting, meteorism, etc.) fail to recur.

The significance of diseases of the middle ear and the mastoid process in children with severe acute gastro-enteritis is still under discussion. Are these real inflammatory diseases which originally participate in the development and continuation of severe diseases, or are they only some of the many complications which occur in the course of a toxic gastro-enteritis? Is the surgical opening of the mastoid process essential for the cure of the disease, even in cases where the finding of otitis media cannot be confirmed, or is it permitted to observe a period of watchful expectancy as long as there are no definite signs of otitis media or of a mastoiditis? While the pathogenetic significance of the disease has been emphasized from some sides and the operation demanded "in every case" for patients with severe symptoms, other experienced physicians are by far more cautious in the evaluation of the pathogenetic significance as well as the surgical treatment of these conditions. In all patients who had died of acute gastro-enteritis, purulent masses are found in the cells of the mastoid process which microscopically turn out frequently to be only mucous secretion and not inflammatory exudate. The problem of the existence of a so-called latent otitis or a latent mastoiditis has not yet been clarified. In any case it appears incorrect to place too great importance in the finding of secretion in the cells of the mastoid process following surgical opening. These findings do not suffice to explain the development of the severe disease and the improvement following the radical operation can only occasionally be considered as the result of the intervention. It seems more likely that these processes are complications of secondary significance which are not decisive for the pathological occurrences. If otoscopic examination reveals the finding of an otitis media or if there are signs of a mastoiditis the therapy which applies to these conditions (antibiotics, paracentesis or mastoidectomy) is of course absolutely indicated. However, as long as there are no definite symptoms and changes surgical intervention should be carried out with caution and reserve or preferably should be omitted.

Provisions for the care of the patient are also included in the treatment of a toxic gastro-enteritis. Considering the many difficulties which can develop in these diseases with their variable courses, hospitalization is usually necessary. In caring for these patients in the private home situations may arise which cannot be handled with the necessary speed (collapse, conditions of shock, necessity of infusions, etc.). But even in the hospital the child should, if at all possible, not be treated in large numbers but should receive individual care. This care does not only consist of the fact that an experienced nurse is placed in charge of the child. Since today the importance of bacteria in the genesis of gastro-enteritis is known, the isolation of the patient has become necessary. Only in this manner it is possible to avoid a spreading of the infection to other patients. By isolating the patient secondary infections can also be kept away from the child who has a low resistance. Only after weeks following a severe gastro-enteritis has the convalescence progressed sufficiently to make a normal care in the hospital or the home possible without danger.

In the process of convalescence from a toxic gastro-enteritis difficulties occur not infrequently. The development of any complication in the course of the disease impairs the prognosis which in any case is serious. Such disturbances in the course of the disease are as follows:

1) The state of intoxication persists despite interruption in food intake. Coma, grey pallor, etc. remain for days while the decreasing weight curve gradually comes to a standstill. The delay in detoxication occurs mainly in young and previously damaged infants particularly during the first months of life and in children with toxic gastro-enteritis where a specific enteral or parenteral infection plays a role in the pathogenesis. The delay in detoxication, if accompanied by a cessation in weight losses and an improvement of the dehydration must not be the cause for deviating from the increases in food intake which, however, must be carried out with particular caution. The return to full consciousness may take place very gradually even after several days.

2) Secondary infections, frequently in form of so-called paravertebral pneumonias or atelectases occur frequently in the first days of convalescence from gastro-enteritis. Renewed temperature elevations caused by the lung involvement are no cause for digressing from the routine treatment as long as there are no signs which speak for the recurrence of the toxic syndrome. If under the influence of secondary infections renewed loss of conscious-

ness and renewed weight decreases occur, as happens not infrequently, it becomes necessary to start again with an interruption in food intake and special dietary therapy. It need not be stressed that this second hunger period represents a serious danger for the usually severely ill child. Today it is possible to overcome this difficult stage with infusion therapy adapted to the condition of the particular case (see earlier). Antibiotics with broad range (chloromycetin, aureomycin, streptomycin combined with sulfa preparations) today cure many secondary infections which in former years caused the death of the infant. Approved therapeutic diets (buttermilk, protein milk) as well as best nursing care are necessary in order to overcome this serious course of a toxic gastro-enteritis.

3) Interruptions in weight gain occur not infrequently at a somewhat later stage of improving toxic gastro-enteritis, at a time when the initial weight losses have been regained. At this time the intracellular and intercellular water losses seem to be compensated again without, however, the damaged organism having regained the ability for new cell formation. The treatment of these conditions of interrupted weight gain is similar to that of acute diarrhea (see p. 372). By adding small quantities of a substance which is rich in protein and whey, or by complementing the diet with vitamins the child begins to gain weight again.

4) Renewed weight drops with stronger diarrhea and apathy can occur even during the first and second week of the disease. This may take place if for instance digressions were made from the routine treatment through careless and excessively rapid increase in food quantities. These disturbances appear most frequently in those forms of gastro-enteritis where the initial weight drops are followed by rapid rises in the weight curve. The rapid establishment of the water balance misleads to too favorable a judgment of the entire condition. The wrong evaluation of this symptom is all the more serious if it occurs in children who have been damaged in their nutritional state, or in hydrolabile children (see p. 514). Particularly in those infants the compensation of the initial water losses takes place very rapidly and gives rise to faulty conclusions. Renewed dietary therapy and infusions are necessary also when symptoms of toxic gastro-enteritis reappear.

Weight losses alone, which lower the body weight again to the initial level, need on the other hand not be the cause for changes in treatment.

5) In some of the infants suffering from intoxication we observe a complete absence of weight increase following the arrest of the weight losses. In these children, who usually are very young, the period of abrupt weight losses is followed by dystrophy and at times a surprisingly rapid development of atrophy. Addition of substances which are rich in protein and whey, preferably perhaps even replacement of part of the artificial food by breast milk, plasma or blood transfusions and in infants beyond the third month of life, addition of vitamin containing food substances or of vitamin preparations (particularly vitamin B and C) cures this progressing aggravation of the nutritional state.

6) Part of the children with toxic gastro-enteritis continue to show weight losses which cannot be brought to a standstill, as well as progressing dehydration; or the weight decreases continue to such a degree that the child reaches a state of severest "decomposition" within a few days. Once a child has reached this stage it is threatened by a number of dangers with regard to nutrition and infections, which make the chances of recovery in these children always very small. Even after days or weeks a minor cause might bring about renewed weight losses and a state of apathy which can no longer be improved even if the food intake is interrupted. The cause for this is probably an auto-intoxication of the organism through products of the endogenous cell decomposition. French authors have described this condition as "self-destruction" or "self-devouring." These conditions have today become extremely rare due to progress in therapy, even in countries where toxic gastro-enteritis occurs not infrequently.

Toxic gastro-enteritis is always a serious disease. This is shown best in statistics from infant wards where until recently, despite the best care and despite the use of all therapeutic measures, about 50% of all infants suffering from toxic gastro-enteritis died. By the widespread application of infusion therapy, by the introduction of chemotherapy and by suitable treatment of dehydration, acidosis and salt losses, the prognosis of the disease has today improved considerably. At present about 8% of the cases of toxic gastro-enteritis are fatal. Infants in bad nutritional and general condition are particularly endangered.

In the beginning of the disease, when stupor, dehydration, acidosis, hypokalemia and shock dominate the picture, it is almost impossible to offer with certainty a judgment regarding the prognosis of the disease. If after an adequate period of starvation and after introduction of drug therapy a turn to the better becomes apparent after about 12 hours, if the child appears more or less detoxicated, a favorable prognosis may be made. Other cases of toxic gastro-enteritis, which initially appear light, show no signs of detoxication following the interruption in food intake. They continue to remain in a toxic condition or the symptoms of apathy, collapse and acidosis fail to disappear completely. This incomplete detoxication occurs particularly in dystrophic and atrophic children whose functionally inferior cells cannot overcome the damage so rapidly or perhaps can no longer overcome it at all. Cases of toxic gastro-enteritis which were caused by specific infections (salmonella, shigella) (see later) show in particular a lack of tendency to rapid detoxication. The prognosis of toxic gastro-enteritis is closely dependent on the circulatory failure. Danger threatens from anoxemia, failure of the peripheral circulation, kidney insufficiency, and central nervous damages which manifest themselves in form of loss of consciousness, convulsions, elevation of intracranial pressure, increase in blood sugar, slight cell increase and increase of the protein fraction in the liquor. The chance of removal of all these disturbances determines the prognosis of the disease. At times days may pass before the child regains consciousness completely. In any case slow detoxication, with an adequate period of interruption in food intake with simultaneous application of all other therapeutic measures (infusions, chemotherapy) points to a particularly severe disease.

But even at the time when the first stage of toxic gastro-enteritis has been overcome and the child seemingly appears on the road to recovery, there is still danger. A number of weeks always pass until the damages to cells and tissues are completely repaired. During the period of convalescence new disturbances can occur from minor causes. New diarrheal disturbances, recurrence of toxic signs and particularly parenteral infections are events which disturb the recovery. Toxic gastro-enteritis is always connected with a considerable loss in the ability to fight off infections of any kind. A few days after the critical drop in temperature in the period of the first weight gains we observe not infrequently new temperature rises which can be caused by pneumonia, otitis, etc. Even in the eutrophic child four to six weeks pass until the patient can be regarded as cured. In dystrophic and atrophic infants two to three months and more are required until the effects of the acute, severe disease have been overcome.

Only rarely does a well cared for and adequately fed infant develop toxic gastro-enteritis. The histories of children with toxic gastro-enteritis show again and again that mistakes have been made in carrying out an adequate nutrition. While in the eutrophic child major errors are required, minimal irregularities suffice in the atrophic infant to bring about severe damage. Since the bad nutritional state was usually caused by earlier acute or subacute diarrheal disturbances, by faulty nutrition or by infections, it seems natural that a history which is full with such incidences finally ends in catastrophy.

It is essential to recognize in time the threat of a toxic gastro-enteritis. In children who are in a good nutritional state it is then still possible to stop the development of severe toxic conditions. In this period of lability, when diarrhea, pallor, lack of appetite, vomiting, slight apathy and restlessness have already developed, any change in nutrition must be carefully considered. During this period any increases or essential changes in diet should be particularly omitted. This places the toxic gastro-enteritis among the avoidable diseases, at least in eutrophic and in the majority of dystrophic infants. Intoxication due to infection which will be discussed in detail in a later chapter, cannot be avoided with similar certainty.

DIARRHEAS WITHOUT ACUTE DISTURBANCES IN THE GENERAL CONDITION OF THE CHILD

Diarrheas without serious involvement of the general condition are seen no less frequently in practice than those diarrheas which are accompanied by disturbances in the general condition. The fact that an infant eliminates frequent, shreddy, greenish, mucous stools, while his general condition is not affected at all or affected very little, is frequently not correctly evaluated with respect to diagnosis and therapy. In order to restore the normal appearance of the stool the physician prescribes prolonged special diets including rice water, strongly diluted milk mixtures, and discontinuation of sugar and fat, without considering the general condition of the child. The wrong interpretation of the "diarrhea" and the corresponding wrong therapy resulted in the many dystrophic and atrophic infants which until 30 to 40 years ago were found in all children's hospitals and died there. But even today the overemphasis on diarrhea as pathological symptom has not disappeared in the practice of pediatrics. We still observe very frequently that for fear of diarrhea infants are kept for a prolonged period of time on a diet, meaning a diet which is insufficient in quantity as well as quality. It cannot be repeated often enough that diarrhea is only a symptom which in itself never determines the necessary therapy. Only those types of diarrhea which are accompanied by disturbances in the general condition, as described in the preceding chapters as symptoms of acute diarrhea or toxic gastro-enteritis, require energetic and active treatment. Cases of diarrhea where signs of impairment of the general condition are lacking permit a period of watchful expectancy. Only in this manner it is possible to avoid harmful hunger periods and damaging changes in nutrition.

Diarrheas without involvement of the general condition are very frequent during infancy. In infants—and this essentially differentiates this period of life from any other age group—any damages which affect the organism cause disturbances in the most sensitive organ system, the gastro-intestinal tract. The gastro-intestinal tract shows almost too sensitive a reaction to all fluctuations in the well being of the child, similar to the seismograph which records distinctly not only local significant earthquakes but also very distant disturbances which are of no local importance. And just as not every earthquake which is registered by the seismograph is sufficient cause for evacuating homes and buildings, not every minor reaction of the child's intestines in form of diarrhea needs to be the cause for instituting radical therapeutic measures. The above mentioned analogy also gives us a certain understanding for the fact that the digestive apparatus can respond to the most diverse stimuli from different localities with always the same abnormal reaction, that is, with diarrhea. The diversity of causes which might produce diarrhea without involving the general condition is shown in the following outline:

Diarrhea without disturbances of the general condition are encountered:

1) in infants during the first weeks of life, in breast fed children as well as bottle fed children, particularly in institutions.

2) In certain types of quantitative hunger, again predominantly in the youngest infants.

3) in a number of qualitative food deficiencies.

4) in dystrophic and atrophic infants who fail to develop satisfactorily.

The justification for setting aside a group of harmless types of diarrhea which do not in any way involve the general condition is shown by the observation in young, well developing breast fed infants. Neither physician nor mother will be worried if a well developing breast fed infant eliminates two to six liquid, greenish, mucous stools per day during the first weeks of life. If the weight increases are satisfactory the time will calmly be awaited, relying on the natural food, when the stools spontaneously become less frequent, which usually occurs around the fourth to sixth week of life. A similar restraint in therapy is customary when the healthy older breast fed infant occasionally develops diarrheal stools without any essential involvement of the general condition of the child.

> This is not a recent discovery. Two hundred years ago *Rosen von Rosenstein* wrote: "The younger we are, the better is our elimination and our sleep; on the other hand, when we get older, the stools get harder and we find less sleep. It must therefore not be immediately considered a diarrhea when a child which sucks well, eliminates three or four times a day."

These experiences in the breast fed child must, however, also be applied to the bottle fed infant. It is of course true that in these cases the borders between harmless diarrhea and diarrheal diseases are less clearly defined—and this constitutes an essential difference between natural and artificial feeding.

DIARRHEA DURING THE FIRST WEEKS OF LIFE (see p. 136)

DIARRHEA OF INFANTS CAUSED BY INADEQUATE DIET

Harmless diarrheas frequently develop in young infants, more rarely in older children, as a result of a quantitatively or qualitatively inadequate food intake (inanition diarrhea, *Finkelstein*). In these cases it is not always a matter of underfeeding the child by giving less than the amounts of calories necessary for the healthy child with good digestion. Losses of nutritive and basic substances in frequent stools can lead to inanition even with an energy quotient of 100–120 calories.

Not every infant responds to hunger with the development of abnormal gastro-intestinal symptoms. The more frequent reaction of the gastro-intestinal tract to hunger is a reduction in stools. Some infants, however, develop a diarrhea as a result of hunger. As any other hungry body cell, the intestinal cells too suffer in their functions. Whether disturbances in the regulation of the bacterial flora in the intestines or a damage to the functions of the intestinal cells themselves are of significance cannot be decided at present. These theories do not offer an explanation why hunger causes at times an infrequent elimination of stools and at other times a diarrhea.

In the hungry breast fed infant whose food is always qualitatively correctly composed the diagnosis of diarrhea due to inanition, or quantitative hunger, is not difficult. By determining the amount taken in during a day the situation can be clarified. In the artificially fed child the diagnosis of a hunger diarrhea due to quantitative deficiencies is more difficult since in such a case also the quality of the food intake can be inadequate. In contrast to diarrheas due to irritation the diagnosis in diarrheas caused by hunger is further made more difficult by the fact that the general condition of hungry children does not remain entirely unaffected as it does in infants with a harmless diarrhea resulting from an irritation of the intestines. Restlessness, but not infrequently also a desire for sleep and signs of discomfort, occasionally vomiting and a slight pallor occur in hungry children. In contrast to diarrheal diseases, we find here a tendency to subnormal temperatures. There are no weight losses. Even after a detailed analysis of the disease there are patients where the decision as to whether it is a diarrhea caused by hunger or an incipient acute diarrhea can be made only on the basis of the child's reaction to food increase or supplementing of the diet (protein, vitamins, fat) (see later, p. 422).

DIARRHEA OF DYSTROPHIC INFANTS

In contrast to the diarrheas observed in young children, we encounter a harmless type of diarrhea also in later months of life. It occurs in children whose nutritional state had already been damaged through earlier faults in nutrition. These are dystrophic or atrophic children in whom the inferiority of the condition of the cells and cell functions manifests itself in occasional diarrheas. These diarrheas, which sooner or later occur in every dystrophic child, never have a sudden onset but begin gradually and after some time are spontaneously replaced by periods with normal stool eliminations. The general condition of the dystrophic infant does not suffer from these periods of diarrhea while with the onset of an acute diarrheal disease an aggravation of the patient's condition can clearly be observed. Appearance and number of stools can resemble those in acute diarrhea. Watery and forcefully expelled stools as found in acute diarrheal diseases are, however, rare.

Dystrophy with occasional diarrheal stools, or dyspeptic dystrophy (Finkelstein) is one of the most difficult chapters in infant nutrition as far as diagnosis and therapy are concerned. In any dystrophic child suffering from diarrhea the physician will bring up the following two questions:

1) Did the diarrhea contribute to the development and continuation of the dystrophic condition, that is, is the diarrhea the cause of the dystrophy?—or

2) Should the diarrhea be regarded only as a harmless secondary symptom of the dystrophy? Does the diarrhea represent nothing but a symptom of dystrophy which should be accorded the same significance as the many other signs of a damaged nutritional state?

The answer to these questions brings serious consequences for the therapy. If the diarrhea is regarded only as a symptom within the sphere of the dystrophy, the aim of the therapy will be to compensate for the errors in nutrition which led to the state of dystrophy. If, on the other hand, it is assumed that the diarrhea is the original cause which led to the development of the dystrophy, the cure of the diarrhea is the first task of therapy. In such cases a special therapeutic diet is necessary which is never without danger for a dystrophic child. Are there any aids which facilitate the correct diagnosis of a diarrhea?

Examination of the stool itself provides no clues. Neither consistency nor color, not even the number of stools are of decisive significance. On the other hand, the behaviour of the weight curve and the influence of the diarrhea on the general condition of the patient offer indications for solving the problem.

As long as the frequent and loose stools do not essentially disturb the progress in weight gain it can justifiably be assumed that the diarrhea is harmless. It is permissible to continue with the entire diet which is necessary for curing the dystrophy, despite the increased peristalsis, or even to supplement or increase the food in quantity and composition. If the weight fails to rise or even shows a decrease which gradually continues, even though the diet is adequate with regard to quantity and quality, it shows that despite a complete diet the functions of the body are disturbed in carrying out the utilization and anabolism of nutritive substances. This indicates the advisability of a special therapeutic diet which should be prescribed as in any acute diarrheal disease.

In patients suffering from diarrhea without any essential involvement of the general condition, the metabolism is only little affected. In cases of harmless diarrhea water retention and water absorption remain entirely undisturbed, in contrast to acute diarrheal disturbances. There are no signs of dehydration, although the water quantities which are lost in the stools of children with harmless diarrhea are no less than those lost in acute diarrhea or in toxic gastro-enteritis. In these cases, however, the shunting of water elimination from kidneys to the intestines suffices to protect the organism from extra- and intracellular water loses. In normal stools the relationship of water in urine to water in stools is 4:1. In harmless diarrhea the stools contain the same large quantities of water as in acute diarrheal disturbances. The quotient of water in urine to water in stools is, however, reversed to 1:2 or even 1:4. More water is eliminated in the feces and less water in the urine.

Otherwise the metabolism of organic and inorganic substances does not seem to be disturbed in any of these types of diarrhea. However, no special investigations have been carried out in this field.

The prognosis of these diarrheas is good and the terminology of "harmless diarrhea" is justified. Damage can result only through inadequate treatment, when the significance of the abnormal stool eliminations is overestimated and superfluous hunger periods are prescribed.

Treatment of Diarrhea without Disturbances of the General Condition

For the majority of patients it is undoubtedly more correct to treat the diarrhea by waiting, that is, without any basic changes in diet, provided the diarrhea appears as the only pathological symptom. The absence of any radical therapy is indicated for those patients with frequent stool eliminations who continue to show an undisturbed satisfactory weight gain. This attitude is of particular importance for diarrheas which occur in young infants in institutions and for diarrheas which accompany a state of dystrophy. Experience shows that these diarrheas cease spontaneously after the child continues to gain weight for several days with a complete diet.

In the introduction to this chapter it has already been emphasized that unsuitable treatment of diarrheas in young infants caused the failure in artificial feeding until not so many years ago. Any minor increase in peristalsis was at that time sufficient indication for introducing a special therapeutic diet, including periods of hunger. The children were exposed to these repeated periods of undernutrition at an age when they were particularly dependent on a complete nutrition and when they especially required at least a constant supply of carbohydrates, water and salts. The many difficulties which were formerly caused by the nutrition of young infants, particularly in institutions caring for infants, have disappeared since the undernourishment of these children has

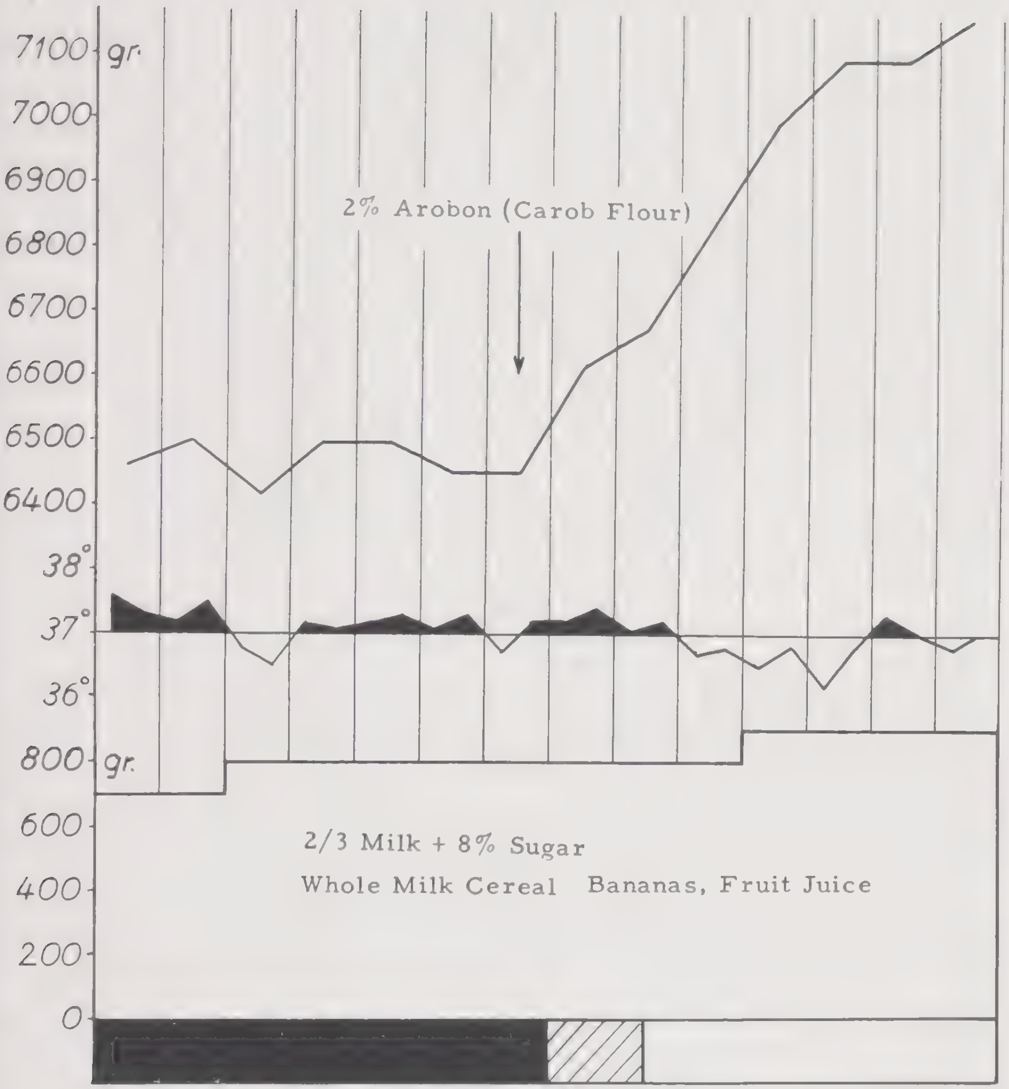

Fig. 47. Infant J.G., five months old. Diarrhea without disturbance of general condition. Cessation of diarrhea and weight increase after addition of 2% Arobon (carob flour) to normal diet.

been avoided. In institutions changes in the diet can be omitted without difficulty. In the private home a "beautifying" of the stools might at times be unavoidable. Even in those cases, however, the basic diet is not changed if it is quantitatively and qualitatively adequate. The following therapeutic means are suitable for improving the appearance of the stools: addition of lactic acid, of calcium bicarbonate, bolus alba steril., bismut

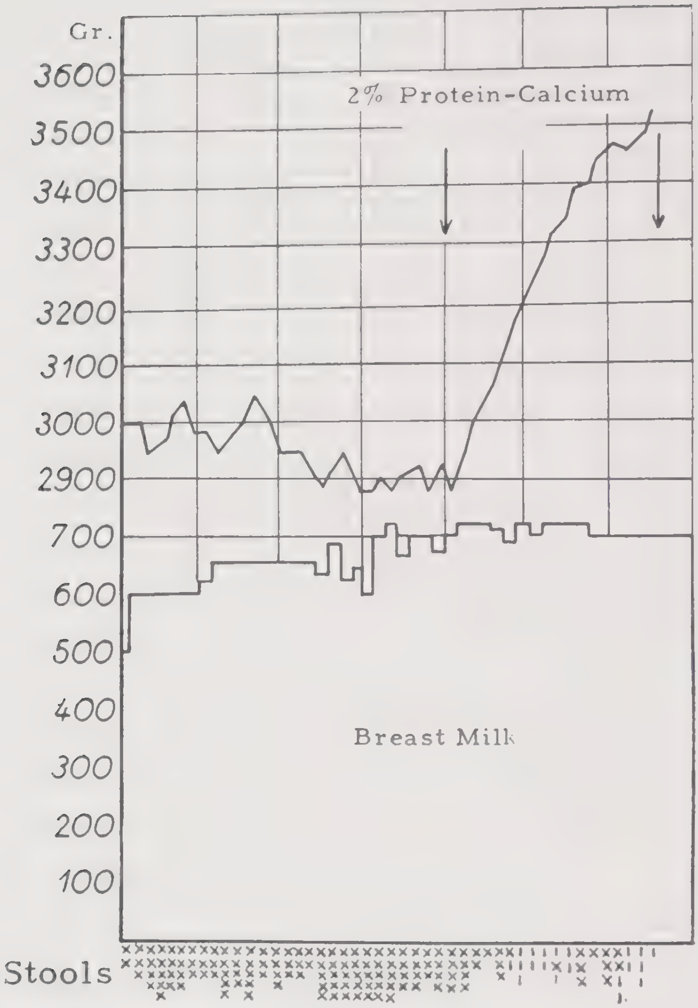

Fig. 48. Dystrophy and diarrhea with adequate nutrition on the breast. Rapid recovery from dystrophy and diarrhea after addition of 3 times 5 g. of a protein preparation.

subgallicum, animal charcoal and others. Carob flour (Arobon, Ceraton), added to the diet in amounts of 2 to 3%, has also been found effective in improving the appearance of the stools. The use of sulfa preparations or antibiotics is not indicated in every case.

Difficulties in the treatment of harmless diarrheas in young infants start only when there is no normal weight gain. In these cases the correct methods of treatment are shown by the experiences gathered in breast fed infants suffering from diarrhea. No physician would replace breast milk by some other type of food only because the number of stool eliminations has increased and because the stools are more liquid. By minor corrections in the diet the number of stools gradually decreases. The same applies to the bottle fed infant. The changes in diet consist essentially in a careful caloric increase, even if the calculated quantities given previously had been calorically adequate, and in the addition of nutritive substances which have a sedative action on the overly active peristalsis.

This aim can be reached in many ways. The food intake can without danger be increased to an energy quotient of about 130. A similar effect can be obtained without increase in food quantity, by adding a protein calcium preparation (Casec or a similar product) which is given in amounts of 1 to 2% of the previously given food quantities. Or concentrated protein milk can be added by giving 30, 50 to 100g per day, distributed in small quantities with every meal.[*] Additions of 10 to 20g powdered milk or buttermilk powder, with the same amounts of sugar, given with the regular diet, or small quantities of condensed milk have a similar effect.

There was in former years a tendency to explain the development of diarrheal eliminations in the artificially fed young child with the unsuitability of heterogenous food. This was sufficient indication for changing over to feeding with breast milk. The physician should not advise against giving the youngest children with frequent diarrheas breast milk in calorically adequate quantities. There is, however, no absolute indication for a transition to natural food.

If breast milk is to be given it is necessary to observe a number of precautions. Because of its composition breast milk is not without danger for children with diarrheal stools. It is therefore not advisable to replace immediately the entire food quantity or a larger part of it with breast milk. More severe diarrhea and weight losses, that is, a diarrheal disease can develop following such a procedure. A temporary special diet is indicated. Tea for four to 6 hours, followed by small quantities of about 300g of breast milk per day, which are then increased from day to day by 100g.

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[*] While regular protein milk is prepared with 10% of protein milk powder in these cases 15 to 20% of milk powder is used in the preparation, with the addition of 7 to 10% sugar.

The results of adequate treatment frequently are surprising. The reaction shows immediately in the weight curve. Already during the first days following the addition of protein to the diet the weight curve begins to rise. The weight increase persists, provided the stools have become normal, if after 10 to 14 days the addition of protein preparations etc. is again discontinued and only the previous food is given in adequate quantities. Frequently the abnormal stools improve only slowly. Only after the weight increase has continued for several days the stools become less frequent. This reaction seems to be proof that the additional substances do not primarily regulate the intestinal processes but that at first the cell functions in the organism are restored. This simple therapeutic procedure, particularly the avoidance of hunger, is one of the conditions which makes the raising of young infants possible also in the mass care of institutions. By accomplishing the continued weight increase of the infant in this manner his immunity and resistance, which formerly were frequently destroyed by the then customary hunger therapy, are also preserved.

Only a small group of young infants with seemingly harmless diarrhea fail to react to the increase or concentration of food with the desired weight increase. A diarrheal disease develops. The weight decreases and the general condition is clearly disturbed. In such cases it is indicated to apply the therapy used in the treatment of acute diarrheal disease.

In treating the frequent stool eliminations which often occur in all dystrophic children, it is a mistake to respond to any recurrence of diarrhea in the course of dystrophy with renewed hunger and special therapeutic diets. Any reduction in diet prevents a cure of the dystrophy. It is incorrect to assume that a condition which has developed due to incomplete diet and hunger can be cured by hunger therapy. In judging and treating the frequent stool eliminations in the dystrophic child, calmness and experience are particularly important. In order to cure the dystrophic condition and thus the diarrhea, it is necessary to prescribe a diet which corresponds to the requirements of the organism and the age of the patient and which is chosen in consideration of the causes which brought about the dystrophy. For all further details we can refer to the chapter on nutritional disturbances.

In treating dystrophic children in institutions where diarrheal stools occur still more frequently than in the private home, it will be necessary to attempt to render the conditions similar to those of individual care by isolating the child, by separating all utensils used for his care, by sterilizing the eating utensils, by frequently washing the hands and by individual attention given to the patient. It is always a mistake to leave a dystrophic child suffering from diarrhea in a state of non-increasing weight for any longer period of time. It is in the interest of the child to force a decision by increasing or complementing the diet in regard to quantity and quality. If increase or supplementation of the diet results in a weight increase of the child the diarrhea will also disappear within a few days. If, on the other hand, the food increase results in a weight loss and in signs which point to a diarrheal disease, the situation changes. The treatment of the child is then the same as in any acute diarrhea or gastro-enteritis. The necessary special therapeutic diet with the rapidly following new dietary increases is more advantageous for the infant than a period of inactive waiting. Even in children where no weight losses occur a prolonged period of non-development and persistent diarrhea represents a serious damage to the child which affects particularly the immunity and resistance of the patient.

CHAPTER IX

Nutritional Disturbances Caused by Infections

ENTERAL INFECTIONS

The enteral infection as cause of acute nutritional disturbances today assumes first place in the pathogenesis of diarrheal diseases. It might be justified to include any diarrheal disease, any acute diarrhea and any toxic gastro-enteritis in this chapter. The invasion of normal intestinal bacteria or bacteria of the paracoli, proteus or pyocyaneus group into the small intestines is today considered the first and essential step in the development of acute diarrheal diseases.

In spite of this it seems at the time still justified to present the acute, more specific infections separately as long as so little definite information is available regarding the biology of "common" germs and as long as their pathogenic significance is not definitely established. In many patients with acute diarrheal diseases it is not possible to prove the presence of a certain well definable bacillus in the stools or even less in the blood, on the basis of the reactions which he produces. We shall therefore describe as enteral infections a number of diseases where either on an epidemiological basis or by the proof of specific bacteria and their products of reaction a definite characterization of the disease is permitted. It should, however, be emphasized already here that in many cases even specific bacteria produce syndromes which to a large extent resemble those which have been described as acute gastroenteritis or as acute diarrhea. Only occasionally one of the symptoms or an unusual reaction toward a therapy which has been found effective in acute diarrheal diseases will lend a specific aspect to the picture of the disease. In addition, specific infectious intestinal diseases such as dysentery or typhoid fever do not always appear during infancy in the known form as described in

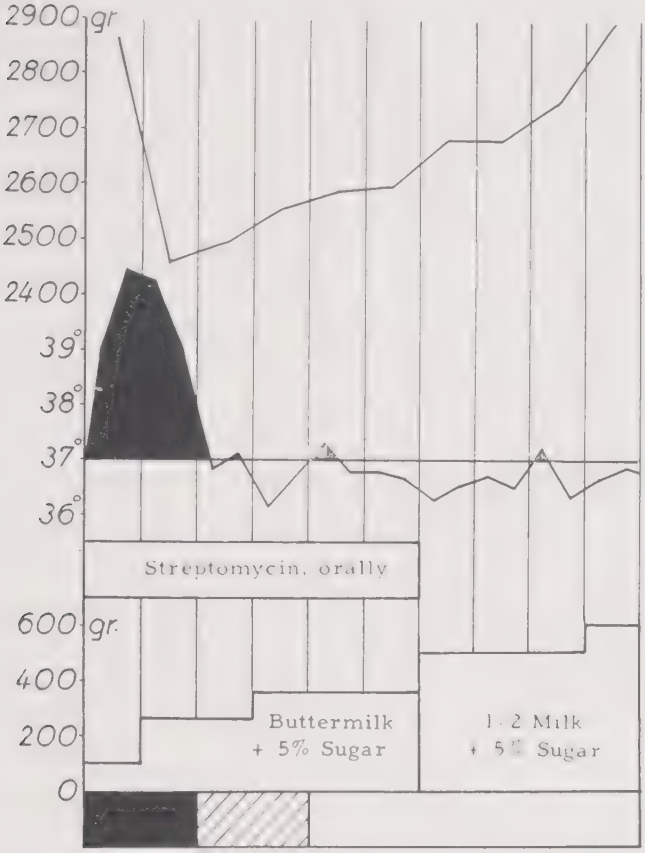


Fig. 49. Infectious diarrhea in the newborn (diet: $^1/_2$ milk with 5% sugar), on the seventh day: fever and diarrhea. Cure with buttermilk and streptomycin per os.

the text books of pathology of the adult. Much more frequently these diseases which are caused by well characterized bacteria hide during infancy behind the mask of an acute or subacute diarrheal disease. Only bacteriological examinations of the stool or the proof of specific products in the blood, the titer of which increases with repeated examinations, reveals the acute diarrheal disease as a bacillary dysentery or as paratyphoid fever. Occasionally it is possible that minor occurrences such as a single bloody stool, particularly abundant admixture of mucus, perhaps also the behaviour of the temperature curve or the appearance of a splenomegaly can direct the suspicion to one or another of the specific intestinal diseases. At other times even these indicative symptoms are absent. Only an almost accidental bacteriological or serological examination, a coincidence of other similar cases uncovers the real nature of the disease. A second reason which permits discussion of the specific infectious intestinal diseases at this time is the treatment of these diseases during infancy. In the young child dietetic treatment of dysentery and paratyphoid fever can even at present not be omitted, despite the introduction of effective drugs. The methods of dietetic treatment are the same as have been found effective in the treatment of acute diarrheal diseases. Only in a few instances the diseases caused by specific intestinal infections require special measures in the dietary procedure.

Among the specific infectious intestinal diseases only the shigella and salmonella infections are of more practical significance for the first year of life. Cases of Typhus abdominalis (typhoid fever) are in many countries extremely rare during infancy. The following discussions are therefore limited to the description of the clinical picture and therapy of dysentery and paratyphoid fever. In infants it seems to be of even less significance than in adults as to which particular species of the large shigella or salmonella family causes the disease in the individual case.

The so-called infectious diarrhea of the newborn should also be included in this discussion because this too is an infectious disease even though the nature of the organism is as yet not determined.

INFECTIOUS DIARRHEA OF THE NEWBORN

Occasionally endemic severe acute diarrheal diseases occur among newborns in a maternity station, which affect particularly those infants who are entirely or partially fed with artificial food. Breast feeding, however, offers no absolute protection against the disease even though the number of breast fed infants affected is only about one half of the artificially fed children. The diseases are not uniform in their etiology. Epidemics of this kind have been caused by salmonella infections in cases where a mother or nurse suffered from a light case of diarrhea which was caused by the specific organism or where they were carriers. Infections can occur due to infected food or infected laundry where the bacteria of the salmonella group can remain alive for a considerable length of time even after the customary washing processes. In other cases the epidemic can be caused by bacteria which in general are not regarded as pathogenic bacilli (e.g., bacillus mucosus, staphylococci, proteus, pyocyaneus). And finally epidemics have been reported where a virus infection has been proved as cause of the disease although it has so far been impossible to prove the presence of a specific virus.

The onset of the disease is insidious. Young infants during the very first days of life, almost always children in the communal care of an institution, also infants who were discharged from the institution to the home before the outbreak of the epidemic, begin to refuse to take their milk. The children cease to gain weight and become restless. They begin to regurgitate and occasionally to vomit. Diarrhea develops, with the stools appearing at first yellowish, mucous, later more greenish. The condition deteriorates very rapidly. The children become sleepy and apathetic. Fever or temperature elevations do not occur in all cases. The children assume a greyish color, the skin becomes dry. With increasing apathy and in complete stupor the children die within 24 hours or a few days. The number of casualties is greatest at the beginning of the epidemic. Gradually the disease becomes somewhat less severe in the afflicted group. Complications on ears (otitis) and lungs (bronchopneumonia) are frequent. Occasionally an acute, infectious lymphadenitis develops below the lower jaw, although no injury of the oral mucosa can be found as source of the lymphadenitis. The incubation period of the disease is short. Diseases occur in intervals of two to three days. But as late as three weeks after discharge from the hospital new cases of the disease have been described in infants who had been among the group of newborns where epidemic diarrhea had occurred. In the different epidemics the mortality rate amounts to 10 to 60% of exposed children. The morbidity rises up to 70%.

The disease is infectious. It spreads from child to child, transmitted perhaps by the hands of mother or nurse. With regard to therapy, streptomycin per os as well as aureomycin and chloromycetin (50 mg per kilogram of body weight, distributed among three to four portions daily) seem to have a favorable influence on the course of the disease. Infusions with salt or glucose solutions, with plasma etc. are necessary in addition to the dietary therapy which corresponds to that applied in acute gastro-enteritis. Among the therapeutic diets buttermilk has been found most effective. A transition to breast milk is desirable.

A newborn department or room where the first cases of diarrhea of the newborn appeared must be closed. The infants suffering from the disease should be taken from the department and strictly isolated. Healthy children should, if possible, be discharged home immediately with the mother.

For prevention of the disease strict nursing routine must be observed in the newborn department. Eating utensils, bottles, nipples, spoons must be sterilized. The use of sterilized laundry is desirable.

Pathologico-anatomical findings in infants who had died of diarrhea of the newborn are completely negative. They have not contributed to the clarification of this acute malignant disease.

BACILLARY DYSENTERY (SHIGELLA INFECTIONS)

In institutions as well as in the private home cases of bacillary dysentery occur every year, usually beginning after the month of June and in fall. A remnant of the so-called summer peak of diarrheal diseases which still appears every year, although in greatly reduced number, is in part the expression of the greater frequency of shigella infections during this season.

The frequency of occurrence in the different years, in different countries and population groups, is closely dependent on the social situation of the population. War, migration, hunger, concentration of people in camps under unfavorable hygienic conditions, are followed rapidly by an increase in the number of dysenteries. Prosperity and favorable hygienic conditions reduce the number of cases of bacillary dysentery.

Dysentery during infancy frequently does not show the typical symptoms of the disease. Abortive and hidden forms are frequent. With the accumulation of cases of intestinal diseases with fever in a group of children the epidemiological correlations reveal very soon the nature of the diseases. In the private home the special type of diarrheal diseases remains undiagnosed until another case of typical dysentery in another member of the family reveals the connection, since the results of bacteriological examinations are very frequently negative when the material is not cultured immediately, if possible already at the bedside of the patient. Serological examination for the presence of specific agglutinins gives positive results only eight to 14 days after the onset of the disease. Negative results of serological examinations are not infrequent even in epidemiologically or bacteriologically proven cases of shigella infections. Also during infancy dysentery is an acute toxic disease. The germs invade the intestines through the mouth with infected food, infected water, through the hands of the nurse, etc. An ascension from the rectum seems to be much rarer. Bacillary dysentery beyond the intestinal wall is found extremely rarely. All the severe and dangerous pathological symptoms which are characteristic for the dysentery of the infant are caused by the toxins of the shigella bacilli which are absorbed through the damaged intestinal wall. This intoxication varies in intensity in the different patients. It can be aggravated to the point of dehydration and toxicosis.

One of the many strains of shigella can be considered as cause of the disease. In general it is not possible to accuse individual strains of causing particularly light or particularly severe cases of the disease. All this depends more on the age of the patient, on his nutritional state and on other as yet unknown factors.

Dysentery starts in the infants mostly from full health, without prodromal diarrhea. Even the eutrophic breast fed child is not protected against dysentery. Dystrophic and atrophic children, particularly also patients with nutritional disturbances (e.g., scurvy) have a predisposition to dysentery and in these children the disease is usually of particular severity.

The symptoms pointing to the presence of dysentery are primarily the stools which contain mucus, pus and streaks or spots of blood. The stools are semi-liquid or liquid and even in the infant are frequently eliminated in smallest quantities, under tormenting tenesmus and colic. But even these symptoms can be missing temporarily or even permanently. The stools of the children then resemble the greenish, mucous, shreddy stools which are eliminated by children suffering from acute diarrhea or toxic gastro-enteritis. The frequency of these pathological stools is, however, not decisive for the prognosis and therapy of dysentery. The course and treatment of the disease are determined by the severity of the general symptoms, as is the case in any other acute diarrheal disease. On the basis of the degree of changes in the general condition three stages of dysentery can be differentiated during infancy:

1) a light form, showing no or only minor disturbances in the general condition aside from the pathological symptoms on the part of the intestinal tract.

2) a moderately severe to severe form, presenting the picture of an infectious or toxic disease with temperature elevations, accompanied by the disturbances in development and in the general condition which are customary in such diseases.

3) the most severe form, resembling poisoning, which progresses rapidly, with hyperpyretic temperatures or apparent lack of fever due to severe collapse.

A transition from a lighter to a more severe form is always possible, particularly during the first days of the disease.

In the light forms of the disease there is no fever or subfebrile irregular temperatures occur following an introductory period with high temperatures. The general condition is hardly disturbed. The children are cheerful and do not even appear ill. There are no gastric symptoms. Accordingly, many children continue to gain weight, particularly when the appetite is little affected or not affected at all. Larger weight losses never occur. The picture resembles most that of a light acute diarrhea which occurred in a well developing child.

The moderately severe cases show the infectious toxic character more clearly. Temperature elevations are not absent. The fever curve is variable. At times the temperatures are irregular, at times they are continuous for days, at times they resemble the temperature curve of typhoid fever, at times they resemble the picture of a septicemia. Gastric symptoms and lack of appetite occur regularly. The general condition is more or less disturbed. Pallor, signs of dehydration, larger weight losses are never absent. In addition, symptoms of acute gastro-enteritis develop in the organism which is damaged by the infection; these disturbances are probably not caused by an ascension of dysentery bacilli into the small intestines. In any case, dysentery bacilli are found in the uppermost sections of the intestines only extremely rarely. On the other hand, non-specific and rarely specific bacilli such as paracoli, proteus, pyocyaneus, etc. are found in the small intestines and even the jejunum of the functionally and anatomically damaged intestinal tract, similar to acute gastro-enteritis. The frequent result of the bacteriological examination in patients with dysentery, "an abnormal flora," can thus be explained. The sensitive shigella flora is apparently easily overgrown by secondarily invading germs. The decision as to whether the toxic signs of dysentery originate from the shigella infection itself or whether secondarily invading germs caused a "non-specific" gastro-enteritis is furnished by the changes of the toxic symptoms following an interruption in food intake. While the symptoms of secondary, acute toxic gastro-enteritis improve with the beginning of a hunger period, the toxic signs caused by the specific toxins of shigella bacilli persist without changes even after the hunger period.

The combination of dysentery and disease of the small intestines presents a serious condition. The spreading of the process to higher sections of the intestines is shown by the changes in the weight curve. In light cases of dysentery, with no weight losses or frequently even continued weight gains, the large intestines can be regarded as exclusive seat of the disease. If larger weight losses occur, an involvement of the small intestines can be assumed. The "poisoning with toxins" then leads to toxicosis. The manifestation of a toxic damage of the nervous apparatus of the intestines is the meteorism which develops in severe cases of dysentery. The appearance of meteorism is a prognostically unfavorable sign.

The most severe form of dysentery is completely dominated by toxic symptoms. These infrequent forms of the disease are the result of an inundation of the organism with shigella-toxins. Most severe symptoms of poisoning, continuous abrupt weight losses, collapse, anuria and symptoms of uremia characterize the

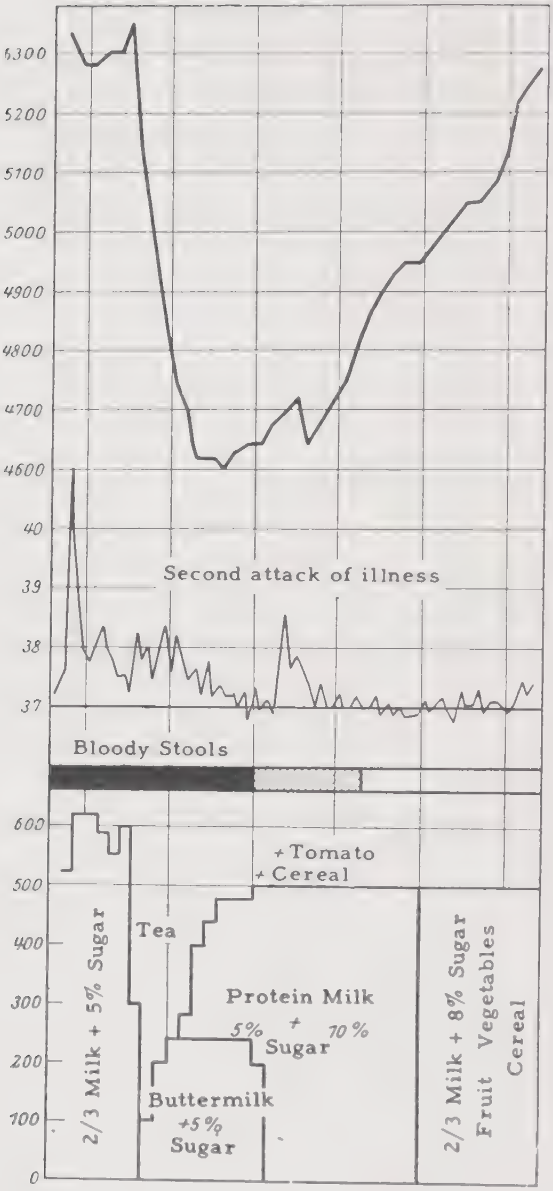


Fig. 50. Acute moderately severe dysentery resembling acute gastro-enteritis. Improvement with buttermilk plus 5% sugar. About 12 days after onset—new fever attack, diarrhea, hemorrhages into the skin, swelling of the joints (second wave of illness).

picture of toxic dysentery. These cases resemble in their course a most severe toxicosis: some patients present the picture of real cholera. A sudden drop of hyperpyretic temperatures to below normal temperatures, without relief of the other serious symptoms and with the pulse rate rising in contrast to the drop in body temperature is a prognostically unfavorable sign. Other children may die with hyperpyretic temperatures and encephalitic symptoms.

The differences in the severity of the disease are in the final analysis caused by the quantities of dysentery toxins, possibly the participation of toxic products of non-specific bacilli which reach the circulation from the intestines, and not by the type of bacillus. An infection with any one of the many bacteria of the shigella group produces in an epidemic light cases and severe cases in equal numbers. It depends on individual conditions to which extent the pathological intestinal process extends beyond the large intestines to involve small intestines and stomach. The spreading of the disease in moderately severe and in severe cases causes serious disturbances in the intermediary processes and leads to dehydration and toxicosis, similar as in any acute gastroenteritis.

The duration of the dysentery varies with the severity of the disease. Light cases can be cured after five to eight days. The majority of cases requires two to three weeks for complete recovery. The toxic dysentery resembling cholera can lead to death within 24 to 48 hours. But even in the cases which recover, a longer period of time is required until the stools return to normal. Slight irritations of the large intestines which manifest themselves in form of admixtures of mucus, of erythrocytes and of leukocytes in the stools frequently persist for many weeks although the child has already resumed his normal appearance.

In a number of infants the overcoming of the first period of fever and diarrhea does not end the disease. Similar to scarlet fever renewed temperature elevations and renewed diarrhea develop during the second or third week of the disease, as well as pathological symptoms which as a whole usually present a light case, at times, however, a severe case or even more serious and severe symptoms than those which had occurred in the first

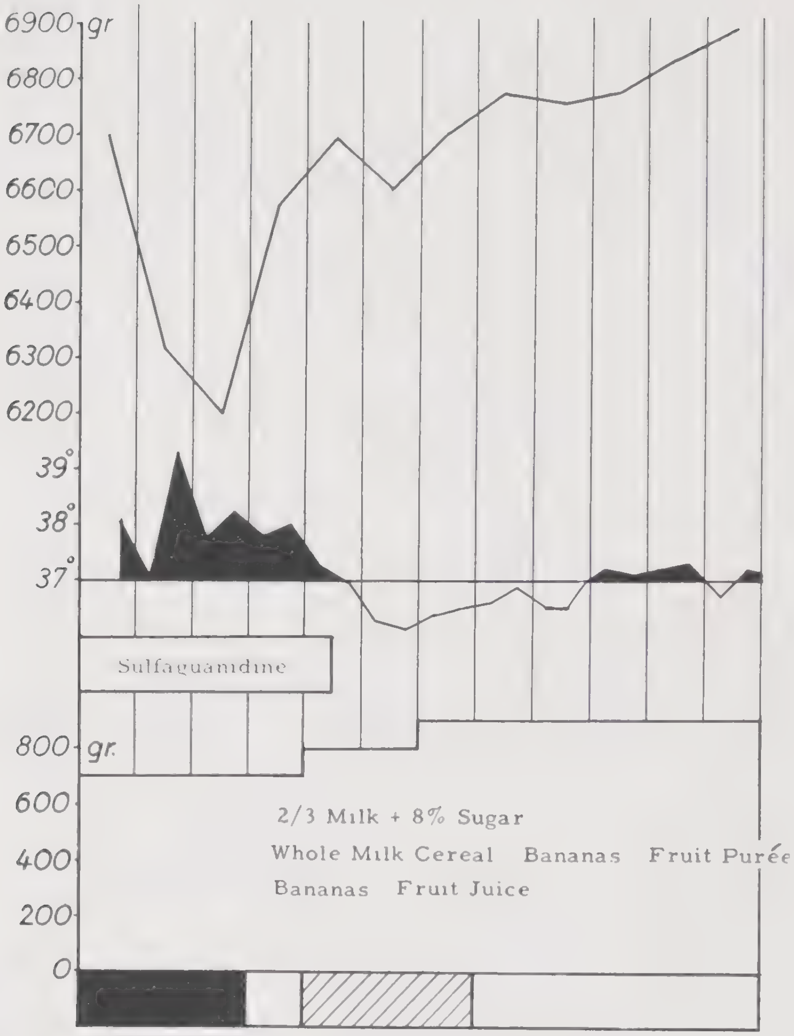


Fig. 51. Infant S.M., eight months old. Dysentery (Shigella Flexner). Moderately severe illness. Cure with sulfaguanidine. Diet: $^2/_3$ milk, cereal with whole milk, fruit purée.

attack of the disease. This recurrence of the disease symptoms is, perhaps not correctly, described as relapse of dysentery and faults in the care or in the diet of the patient are considered responsible for their development. This second wave of illness has been described as *Reiter's* syndrome. At times even a third attack of the disease occurs. The second attack of dysentery usually takes a monotonous course in the infant without serious complications. While the small child develops such secondary symptoms as

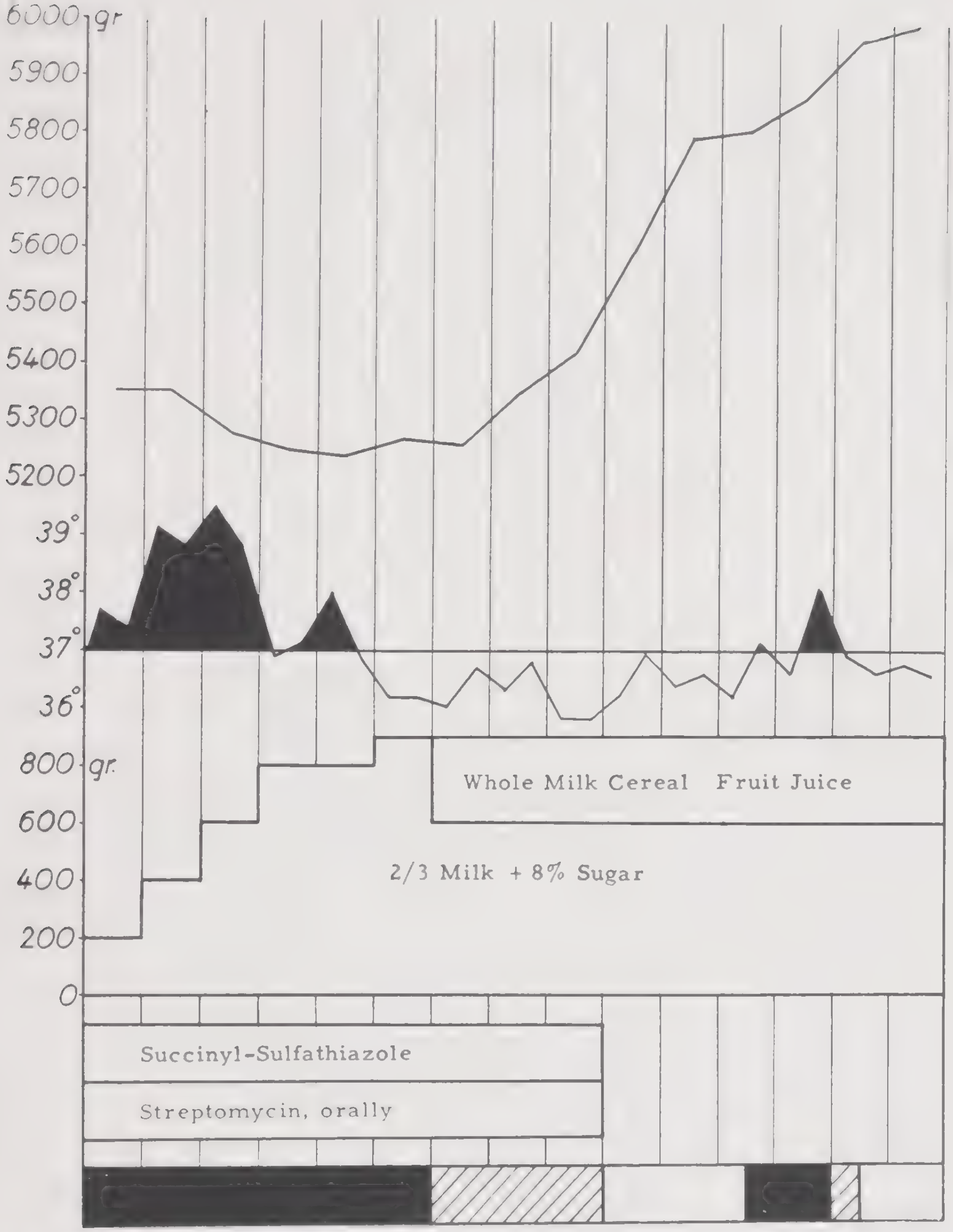


Fig. 52. Infant, four and one-half months old. Moderately severe dysentery. Weight loss and fever, not caused by alimentary indiscretion because there was neither a critical drop in fever nor in weight increase after 12 hours of diet consisting only of tea. In spite of persistent signs of light dysentery, intoxication and continued diarrheas the diet was increased cereal and fruit juices were added early. Rapid cure by avoiding harmful and unnecessary starvation. At the end of the second week re-appearance of symptoms (second illness).

conjunctivitis, iritis, urethritis and joint and gland swellings accompanying the second attack of dysentery, the infant shows only temperature elevations and renewed diarrhea. Occasionally we also find symptoms of a rapidly receding pyuria.

The prognosis of dysentery during infancy is favorable for the light and moderately severe cases. The rarely occurring severe toxic cases of dysentery, however, are frequently hopeless because of their violent course, even under the best treatment.

Before the physician decides on a diagnosis of bacillary "dysentery" or of a shigella infection certain differential diagnostic considerations should be taken into account, particularly during infancy. The elimination of stools which contain blood does not, in any case, suffice for a diagnosis of "bacillary dysentery." Salmonella infections also at times cause bloody mucous diarrhea. In infants the not infrequent intestinal obstructions due to intussusception (see p. 212) are of importance in the differential diagnosis where the syndrome of vomiting, sensitivity of the abdomen and the elimination of bloody liquids or of stools admixed with streaks of blood can lead to an erroneous conclusion. The sudden onset, the absence of fever at least during the first hours of the disease, the severe collapse and the absence of mucus and pus in the eliminations speak in favor of an invagination. The finding of a palpable tumor in the abdomen and localized, visible intestinal contractions are characteristic for a diagnosis of "ileus." Even these symptoms can deceive. Occasionally stool eliminations in children suffering from dysentery cease suddenly or the disease sets in with meteorism and constipation. This sudden ceasing of eliminations is the result of a paralytic ileus which can resemble closely the picture of intussusception. Visible intestinal cramps occur in the patients. The abdomen is filled with gas and distended. The child does not eliminate stools. Vomiting occurs. The roentgen picture reveals over-distended intestinal loops filled with air as well as fluid levels. Occasionally only an operation can resolve the question as to whether it is a case of pseudo-ileus with an acute intestinal infection or a case of invagination (see p. 383).

In scurvy, too, hemorrhages from the intestinal mucosa can develop which then simulate the appearance of a dysenteric stool.

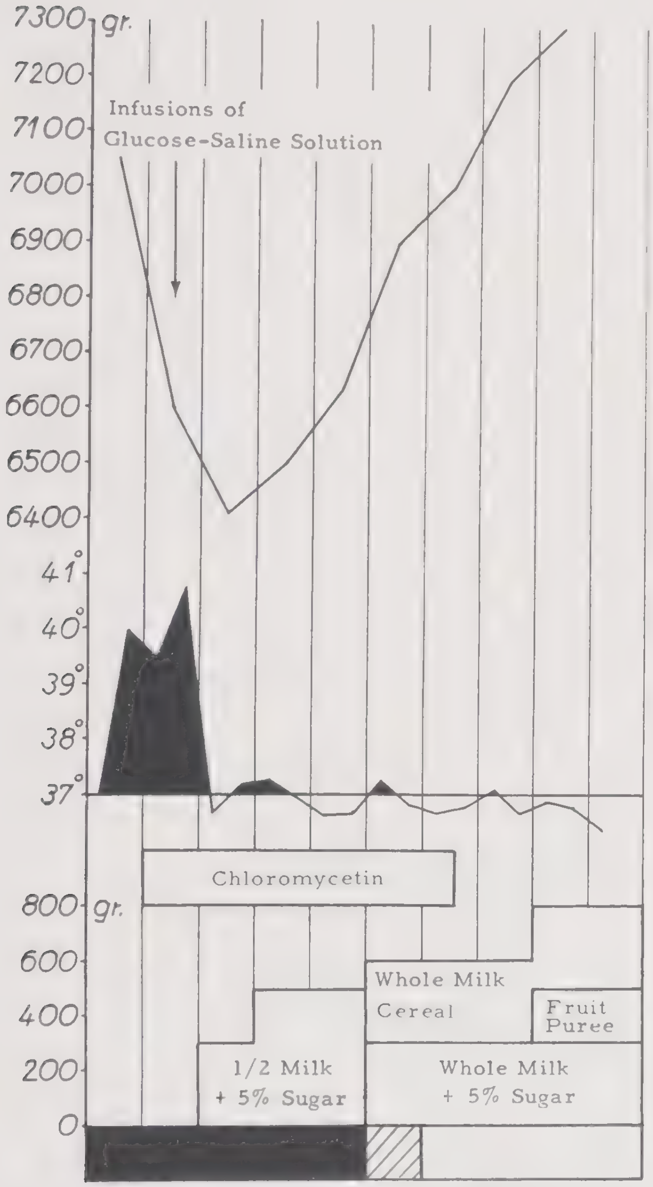


Fig. 53. Infant D.S., eight months old. Dysentery (Shigella Flexner). Severe toxic manifestations, collapse, convulsions. Two days only sugar water. After detoxication recovery with $^1/_2$ milk plus 5% sugar and early change to normal diet.

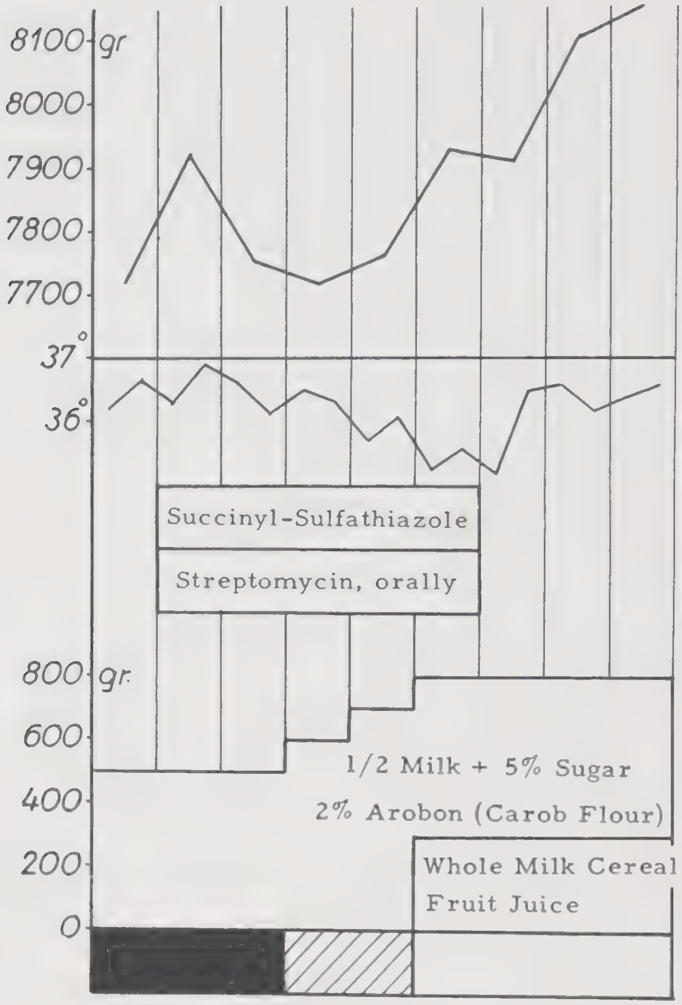


Fig. 54. Infant L.A., 10 months old. Dysentery (Shigella Flexner). Cure with Arobon (carob flour), after two days $^1/_2$ milk plus 5% sugar plus 2% carob flour. Succinylsulfathiazol plus streptomycin per os.

Pus is always absent in the stools of these patients but the fact must not be overlooked, as has already been mentioned, that scurvy in particular is one of the diseases which especially favor infections with shigella bacteria. On the other hand a prolonged case of dysentery can lead to such an increase in vitamin C consumption that scorbutic symptoms are not infrequent in the course of cases of prolonged dysentery.

The pathologico-anatomical changes in the intestines are, even in severe cases of dysentery, frequently less pronounced in the

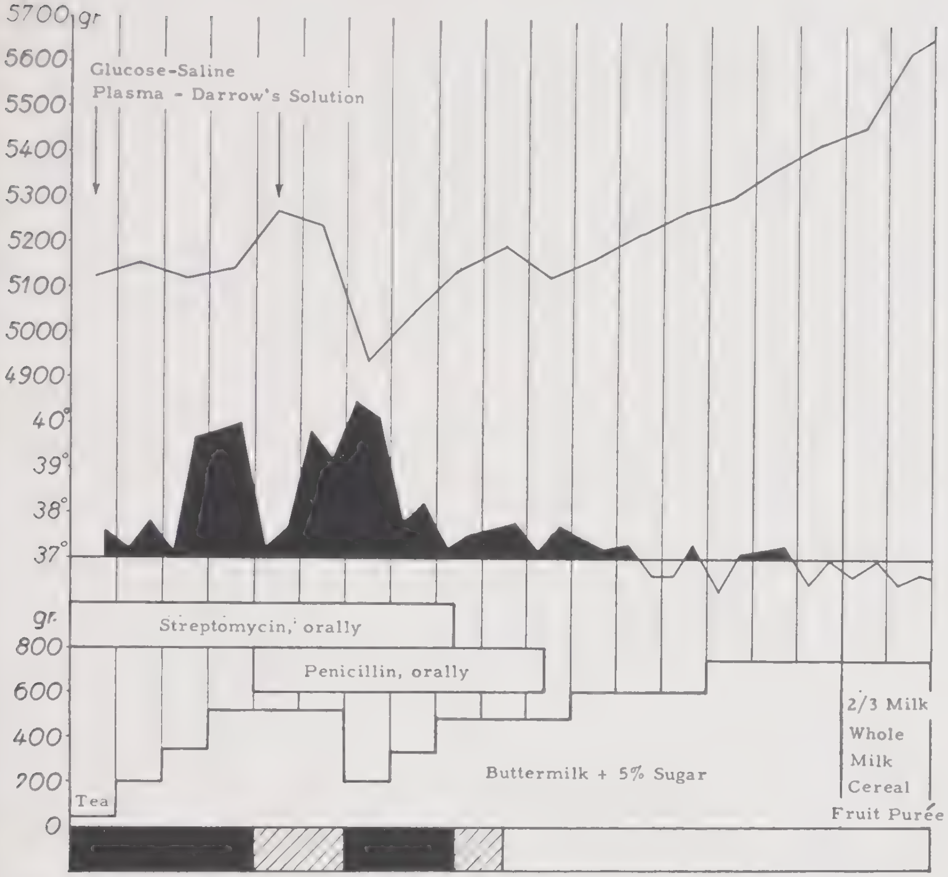


Fig. 54a. Infant J.S., nine months old. Dysentery (Shigella Flexner). Moderately severe illness which improves rapidly with penicillin and streptomycin with $^2/_3$ milk plus 8% sugar in increasing amounts. Second illness with fever and loose stools.

infant than the impressive pathological changes which dysentery produces in the intestines of the adult. Frequently infants who had died of dysentery reveal only a peach-colored discoloration of the serosa of small and large intestines, a moderate swelling of the follicles of the lower intestinal sections and a white soft swelling of the mesenteric glands. The findings of a typical necrotizing inflammation of the large intestines, whereby the intestinal wall appears rigid and thickened and permeated with hemorrhages and infiltrations occurs much more rarely. On the other hand a distension of the lungs is found regularly and can be proved clinically as manifestation of poisoning with toxins. Edema of the meninges and swelling of parenchymatous organs are frequently observed.

The treatment of dysentery has been facilitated considerably by the introduction of sulfa preparations and antibiotics. Despite this progress the dietetic treatment must not be neglected. Dietetic treatment of bacillary dysentery depends during infancy on the severity of the disease. It is incorrect to prescribe for every case of dysentery in infants and young children a severe therapeutic diet which always means a hunger diet, lasting for many hours and days. As in any diarrheal disease during infancy, prolonged hunger is the most undesirable method of treatment also in cases of dysentery.

In light cases of dysentery any basic changes in nutrition should be avoided since the disease consists merely of a pathological process which is confined to the large intestines. The vital digesting and absorbing intestinal sections are not affected by the disease. There is no reason whatsoever for prescribing any strict therapeutic diet in such cases. In younger infants who do not as yet receive any additional food the diet of natural or artificial milk feeding is continued without any changes. If the child receives fat in form of butter or roasted butter-flour mixtures these foods should be omitted. The sugar intake, on the other hand, need not be reduced and it is wrong to omit sugar entirely from the diet. The addition of cereal prepared with farina is also permitted. In children beyond the third month of life it is necessary to continue the supply of 50 to 80cc of fresh fruit juice per day in order to provide the increased vitamin C requirements needed during the dysentery. These amounts of fruit juices do not produce diarrhea. In older children who already receive vegetables the vegetable meal should be replaced by a second dish of cereal, in the child beyond the ninth month of life by rice with the addition of puréed meat or fish. In older children dark bread is omitted for a while in order to avoid the passing of larger quantities of cellulose into the large intestines. If children suffering from a light case of dysentery drink badly the customary milk dilutions can be replaced by concentrated feeding mixtures, e.g., whole milk with 17% sugar, or less diluted sweetened condensed milk, in order to meet the necessary food requirements of the child.

In moderately severe cases of dysentery the dietetic therapy

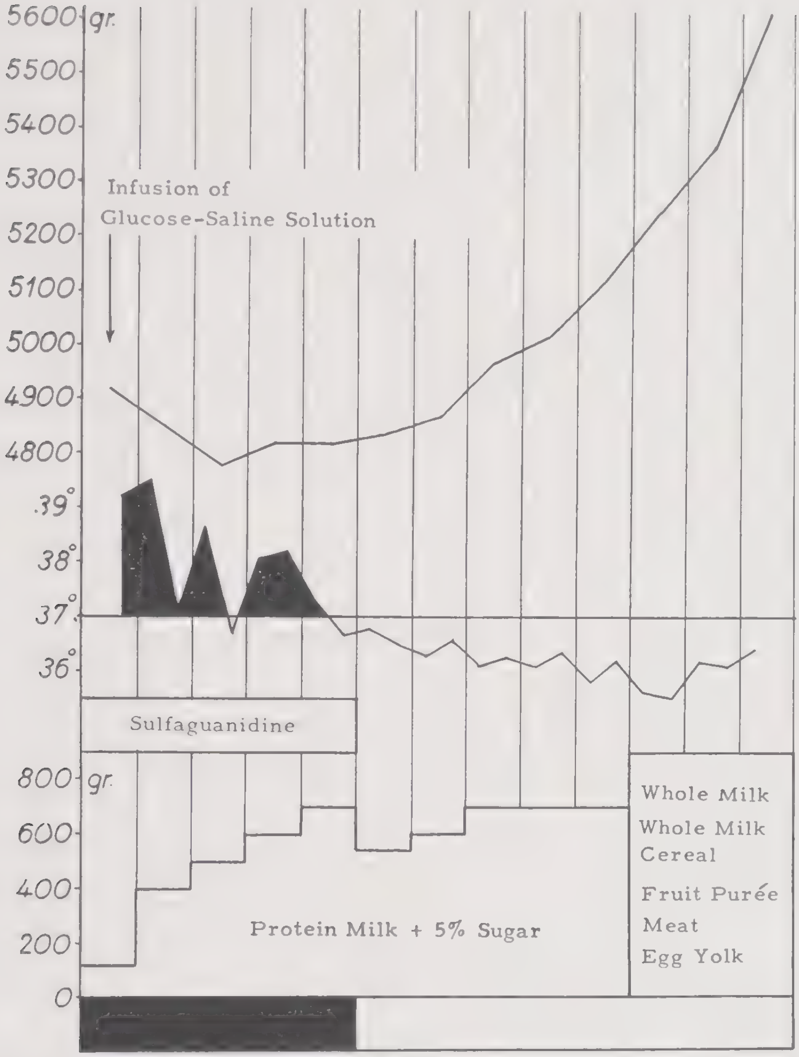


Fig. 55. Child W.N., two years old, weight: 4800 g. Severe dysentery (Shigella Boyd) with severe dystrophy. Cure with diet of protein milk plus 5% sugar. Sulfaguanidine. Discharge weight: 7300 g.

will depend on the extent to which the general condition is involved. In case of fever and weight losses the treatment applying to acute diarrheal diseases should be applied. However, a decision must be made as to whether a treatment suffices which is adequate for acute dyspepsia or whether dietetic measures used in the treatment of toxic gastro-enteritis are necessary. In any case, no food is offered for six to 12 hours, only water or tea sweetened with saccharine, at the rate of 500 to 1000cc per day. In older infants sugar water (10 to 20%) can also be given. The quantities of food given after this hunger period and the speed with which the amounts of food are increased from day to day depend on the reactions observed after the brief interruption in food intake. If the symptoms of dehydration and toxicosis are improved feeding can be resumed with a daily amount of 200 to 300g. Without regard to any bloody mucous stools which might still occur the food quantity can be increased by 100g, in cases with minor general disturbances every day, in cases with more severe disturbances of the general condition at the rate of every other day. It is desirable to add at the earliest possible moment the initially lacking substances such as fruit juices to the diet and to return as soon as possible to a qualitatively correctly composed diet. If, on the other hand, the hunger period does not result in a detoxication and if the weight losses persist, indicating that the specific toxic components of dysentery or of a secondary non-specific infection of the small intestines are still active, more caution is advisable in the increase of food in quantity and variety. In such cases, too, we can refer to the methods as described for non-specific acute diarrheal diseases. The same mixtures which were recommended for the treatment of acute diarrhea have been found effective as therapeutic diets for moderately severe cases of dysentery. These are mixtures composed of buttermilk with 5% sugar, protein milk with 5 to 10% sugar or half-skimmed milk with sugar. In more severe cases the therapeutic diet can be replaced after one to two weeks by the customary milk mixtures even if the stools have by then not returned to normal.

In light cases of dysentery in breast fed children nursing is continued without any changes.. In moderately severe cases with stronger toxic symptoms and greater weight loss an omission of two to three meals can be followed by a careful feeding with small quantities of expressed breast milk (20 to 50 g per meal), five to six times daily. The quantities are gradually increased. After three to four days the child can again be placed on the breast, at the beginning perhaps for a shorter period of time (five to seven minutes). Only in the rare cases where breast fed infants show severe dehydration breast milk will have to be discontinued altogether for the first days of the disease and after a hunger period during which only tea is given, the treatment is started with skimmed breast milk in amounts of 200 to 300g per day or with buttermilk. However, these feeding mixtures are only given for a short time. After a few days the return to breast feeding is indicated.

The dietetic treatment of severe toxic cases of dysentery is far from easy. Any interruption in food intake, even for 18 to 24 hours or more, does not essentially change the toxic condition of these children. As long as toxic symptoms persist no food is given per mouth. Only after the vomiting has ceased and the child no longer refuses the food frequent small meals, for instance 8 × 30g daily of a therapeutic diet, can be offered.

An attempt must be made at all costs to compensate for the severe losses of fluid during the first critical period of this dangerous condition. Dehydration and acidosis which might also exist must be combatted by means of intravenous or intraossal infusions (see p. 394). Infusion therapy with saline solutions, plasma, amino acid hydrolysates as well as blood transfusions should be continued as long as vomiting and pronounced lack of appetite persist. By means of continuous drip infusions or preferably perhaps by repeated infusions it is today possible to maintain the weight of infants even for many days without oral food intake. For the most severe cases of dysentery with their exceedingly large acute weight losses subcutaneous infusion, with the addition of hyaluronidase, can combat the primary great danger of cell damages due to water and salt losses. In case of acidosis which can be diagnosed by clinical symptoms and the results of alkali reserve determinations in the blood, such substances as Hartmann's or Darrow's solution, which are effective against acidosis, should be added to the saline and glucose solutions. Vitamin C and Riboflavin—preferably not simultaneously but in separate portions—should be added to the solutions which are used for infusion. Severe toxic cases of dysentery can be cured only when a compensation for the dehydration and balance of the disturbed salt metabolism are successful and water fixation can be restored.

The drug therapy of dysentery has been considerably enriched.

With the administration of sulfa preparations which are slowly absorbed in the intestines (sulfaguanidine, succinylsulfathiazole, etc.) the development of shigella bacteria in the intestines is arrested. The production and absorption of toxins is reduced or stopped. The same applies for antibiotics which are effective against gram-negative bacteria. This includes streptomycin, aureomycin, chloromycetin, terramycin, etc. Sulfa preparations should be given in quantities of not less than 0.3g per kilogram of body weight per day, distributed among four to six portions. Antibiotics are given to infants in the following amounts: streptomycin 0.25 to 0.5g, three to four times daily, chloromycetin 50 mg, aureomycin 30 mg per kilogram of body weight. The total daily quantity is distributed among three to four portions and given at regular intervals during the 24 hour period. The combination of a slowly absorbing sulfa preparation with an antibiotic (e.g., succinylsulfathiazole plus streptomycin) seems effective. If no definite improvement of the stools can be observed after four to five days it is not advisable to continue any further with the same drug. A change of the drugs seems indicated. On the other hand the medication of sulfa preparations and antibiotics can be discontinued after the stools have been normal for two to three days.

In severe and critical cases of dysentery the treatment of shock and collapse follows the rules applying to toxic gastro-enteritis. Meteorism which is due to toxic influences and paralytic ileus, with an accumulation of bilious and hemorrhagic fluids in the stomach caused by stasis from the intestines is treated by interrupting all food intake and introducing a stomach tube. The stomach tube must remain in place until water which is injected through the tube and then aspirated flows back clear; this may take hours or even days. Injection of prostigmine (0.1 to 0.2 mg) in intervals of four to six hours helps in restoring the peristalsis back to normal.

Among all other drugs only those are indicated in the acute state of dysentery which relieve the symptomatic disturbances of the patient. Atropine and Belladonna are effective for this purpose. In not too small dosages these drugs succeed in bringing relief from the painful colic and the tormenting tenesmus of the patients. No success can be expected from cathartics, not even during the very first hours of the disease. Even at the very onset of the disease the prescribing of castor oil is superfluous or even harmful. Intestinal irrigations of any type should be avoided in infants suffering from dysentery. The recommended constipating substances (animal charcoal, bolus alba, bismuth, calcium, etc.) are ineffective in the acute stage of the disease. Only after the disease is essentially cured and only irritations of the large intestines prevent the formation of normal stools this remnant of dysentery can be improved with the aid of one of the constipating agents. The use of carob flour preparations (Arabon, etc.) is preferable in such cases to the other medications. The therapeutic effect of serum therapy in dysentery during infancy is problematical. The use of antiserum in dysentery has therefore been abandoned.

Dysentery is one of those diseases where judicious and appropriate care is of decisive significance for the course of the disease. Adequate heat supply is important since every child suffering from dysentery shows particular sensitivity to chilling. Particular attention in the care of the skin is necessary in the presence of the frequent stools.

PARATYPHOID FEVER (SALMONELLA INFECTIONS)

The number of cases of paratyphoid fever has been on the increase in many countries. The knowledge of the existence of the tremendous reservoirs of the bacteria of the salmonella group in humans and animals and the improvements of bacteriological and serological diagnosis make it understandable that many diseases which were formerly considered to be acute diarrhea or acute gastro-enteritis of non-specific etiology are today recognized, on the basis of bacteriological or serological findings, as diseases caused by one of the many bacteria of the salmonella group. The different species of salmonella do not cause different symptoms. Light or severe cases, diseases with a course similar to typhoid and short, febrile diseases with or without diarrhea may be caused by any one of the different bacteria. Even in individual epidemics where the same species can be found the form of the disease varies in the different patients. In a small group of children or even in the individual child we find not infrequently not just one but several strains of the salmonella group. Epidemiological research has so far not found any satisfactory explanation for this fact which has been observed repeatedly. The assumption that mixed infections and multiple infections are not rare because of the wide dispersion of the different salmonella species is not very satisfactory. Perhaps the settling of other salmonella strains is easier in a damaged intestinal tract than in the healthy intestines which do not permit their settling and propagation. In view of the many opportunities which exist for such an infection the physician is obliged to consider a salmonella infection in every case of acute diarrheal disturbance.

Although in many patients only the results of bacteriological and serological examinations can provide the final answer there are always some signs among the symptoms which direct the suspicion to the presence of a specific infection with bacteria of the salmonella group. Symptoms which speak for a salmonella infection as cause of a diarrheal disease are the following:

1) Irregular temperature elevations which are not influenced by an interruption or a reduction in food intake and for which no other explanation, such as some other parenteral infection, can be found.

2) The preponderance of gastric symptoms (lack of appetite, vomiting, brownish felt-like coated dry tongue) which do not show the rapid improvement following the customary therapeutic diet as occurs in other acute diarrheal diseases.

3) The tendency to recurrences. After the first symptoms of acute diarrhea or gastro-enteritis have already shown improvement renewed relapses occur although no special reason and no faults in the therapeutic processes can be found. Diarrhea recurs, gastric symptoms increase and renewed weight losses develop.

4) The tendency to many types of complications. Encephalitic symptoms which accompany hyperpyretic temperatures are particularly dangerous. Meningitis, osteomyelitis, bronchitis, pneumonia, pleuritis, whereby the same bacteria are found as appeared in the stools, are not infrequent.

Findings in the white blood picture, on the other hand, provide no aid in the diagnosis. Leukocyte values of 25,000 to 30,000 are found as frequently as leukopenia. Lymphocytosis does not

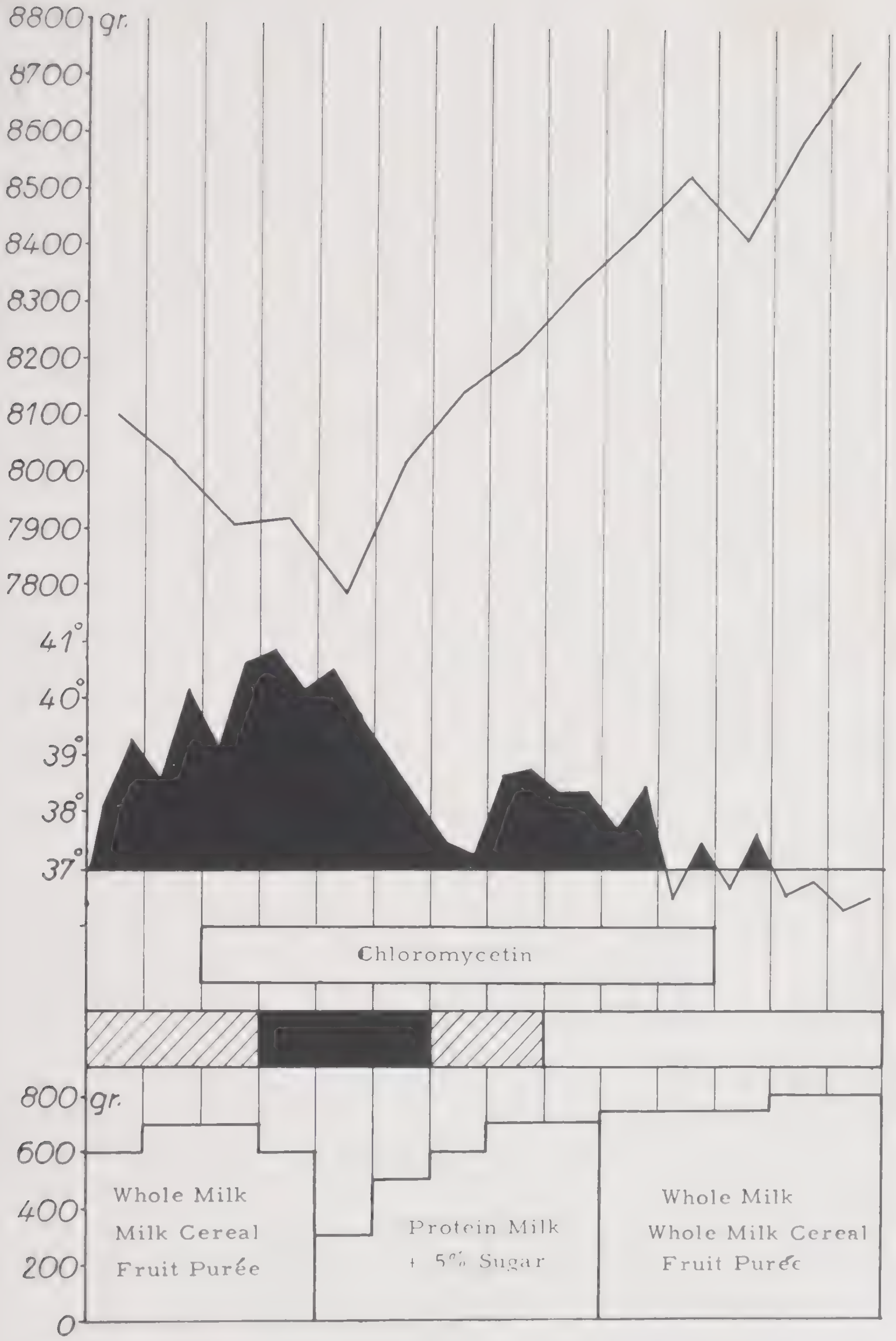


Fig. 56. Infant B.A., eight months old. Paratyphoid (Salmonella B) with symptoms of acute diarrhea. Cure with chloromycetin and protein milk plus 5% sugar.

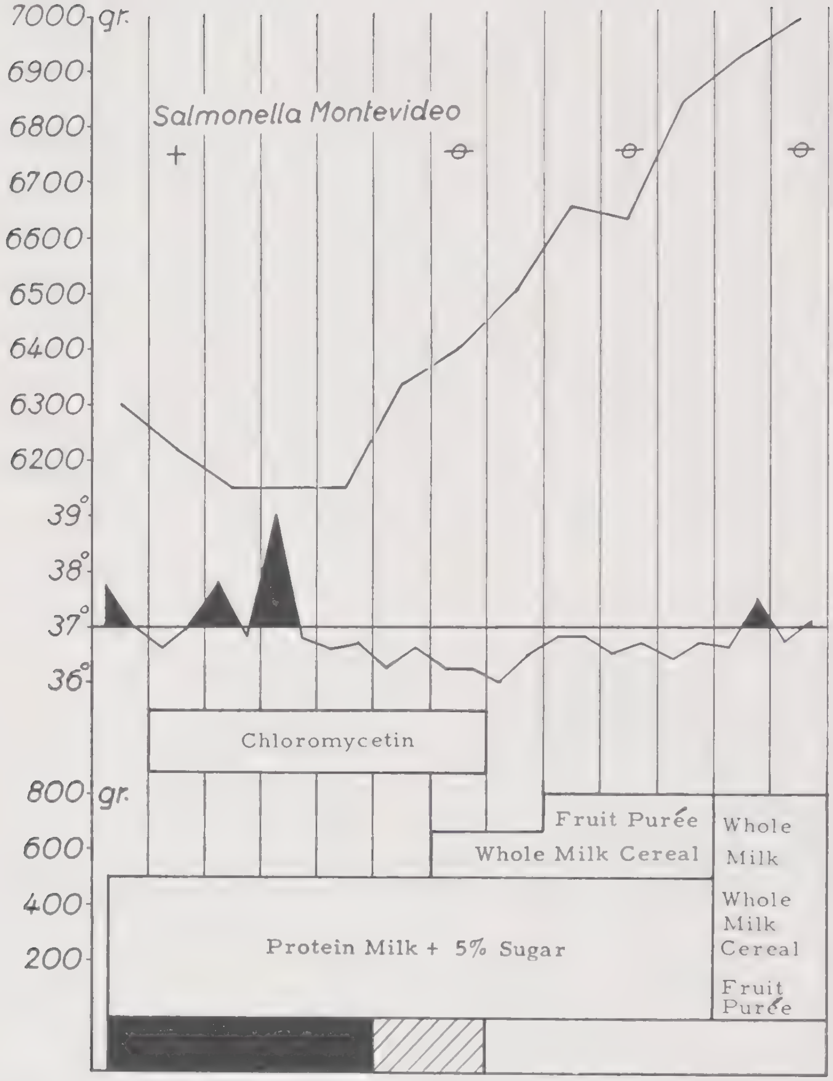


Fig. 57. Light case of paratyphoid fever with symptoms of acute diarrhea. Cure with protein milk plus 5% sugar and chloromycetin.

occur in all cases. Roseola in form of small and large spots can be present at times. In contrast to typhoid fever this exanthema usually is not limited to the area of the abdomen. Roseola is found also on chest, back and extremities. In other patients and particularly in young children roseola does not occur throughout the entire period of the disease. Enlargement of the spleen is not present in all cases. At times the spleen can just be palpated. Marked enlargement of the spleen is rare. The dry, greyishbrown coated tongue is striking, with the tip and the edges of the tongue remaining unaffected by the changes. The children are very weak, greyish, and the eyes appear haloed. Signs of dehydration are frequent due to the violent diarrhea and the violent and frequent vomiting as well as the lack of appetite. Symptoms of avitaminosis caused by vitamin B and C deficiencies develop rapidly. In general young children suffering from a salmonella infection frequently appear severely ill. Apart from these individual cases which on the basis of various symptoms are characterized as something unusual there are, however, great numbers of infections caused by bacteria of the salmonella group the course of which is entirely similar to that of acute diarrhea or toxic gastro-enteritis. In a third group of infants we find the picture of a febrile disease but without pathological symptoms on the part of the gastro-intestinal tract, without diarrhea and without gastric symptoms. In these patients the specific etiology of the disease can be proved only by the finding of the bacteria in the stools and after 10 to 14 days or at times even later, by the development of positive serological reactions. Incorrect preservation of the material which is sent for examination, great distances and delays before the stool is brought for culture cause negative results either because the salmonella bacteria are killed or because they are overgrown by other germs. At times, when the customary methods of examination fail, the examination of the bone marrow might still give a positive culture or agglutination reaction.

Dietetic treatment of patients suffering from paratyphoid fever should be adapted to the form of the disease. Patients with fever but without essential disturbances on the part of the gastro-intestinal tract are best treated with a diet which is poor in undigestible bulk. The food should not be too fat. In older infants substances which are rich in cellulose such as dark bread, vegetables, etc. should be avoided. Protein should be given in quantities of 2 to 3g per kilogram of body weight, particularly for those patients with prolonged high fever.

The form of disease which resembles typhoid fever occurs almost exclusively in children at the end of the first year or beyond the first year of life. In young infants salmonella infections resemble more the picture of an acute diarrheal disease. Here, too—as has already been stressed in shigella infections—it is at times difficult to decide whether the infection of the small intestines occurred primarily through the specific bacteria of the salmonella group or whether other non-specific germs invaded the damaged intestines as a secondary process. In these cases dietetic treatment follows the rules applying to non-specific intestinal diseases with consideration of the general condition of the patient. Dehydration and metabolic acidosis occur also in salmonella infections and must be considered in the treatment. In case of salmonella infections three kinds of difficulties arise with regard to therapy: 1) recovery from diarrhea is slower than in non-specific diarrheal disturbances; 2) the severe gastric symptoms require a prolonged hunger period and frequently and over longer periods of time small frequent meals with a more gradual increase in food quantities in the individual meals; 3) repeated continuous drip infusions are often necessary in severely ill children and those who are ill for a longer period of time, in order to meet the fluid, salt and protein requirements.

Dietetic treatment of the most frequent form of the disease as it occurs in older infants and small children, with irregular temperatures, with its periods of diarrhea followed by periods of normal or almost normal stools, with its tendency to vomiting and to relapses with renewed diarrhea and renewed attacks of vomiting, is extremely difficult. If at the onset or in the course of the relapse renewed diarrhea occurs, accompanied by general symptoms, particularly marked and prolonged weight losses or signs of dehydration and toxicosis, a renewed period of hunger and a renewed reduction in food intake is indicated. These repeated periods of quantitatively and qualitatively incomplete nutrition can today be overcome by adequate parenteral feeding. Care must be taken that the most important vitamins, particularly vitamin C and Riboflavin, are added to the infusion liquid or to the parenteral injection. The regular medication of sodium luminal one to three times daily in not too small quantities is at times effective against the tendency to vomit. Feeding by mouth must, however, always be delayed until the gastric symptoms disappear. In other patients with normal stools and severe gastric symptoms weight losses occur which are treated with a special

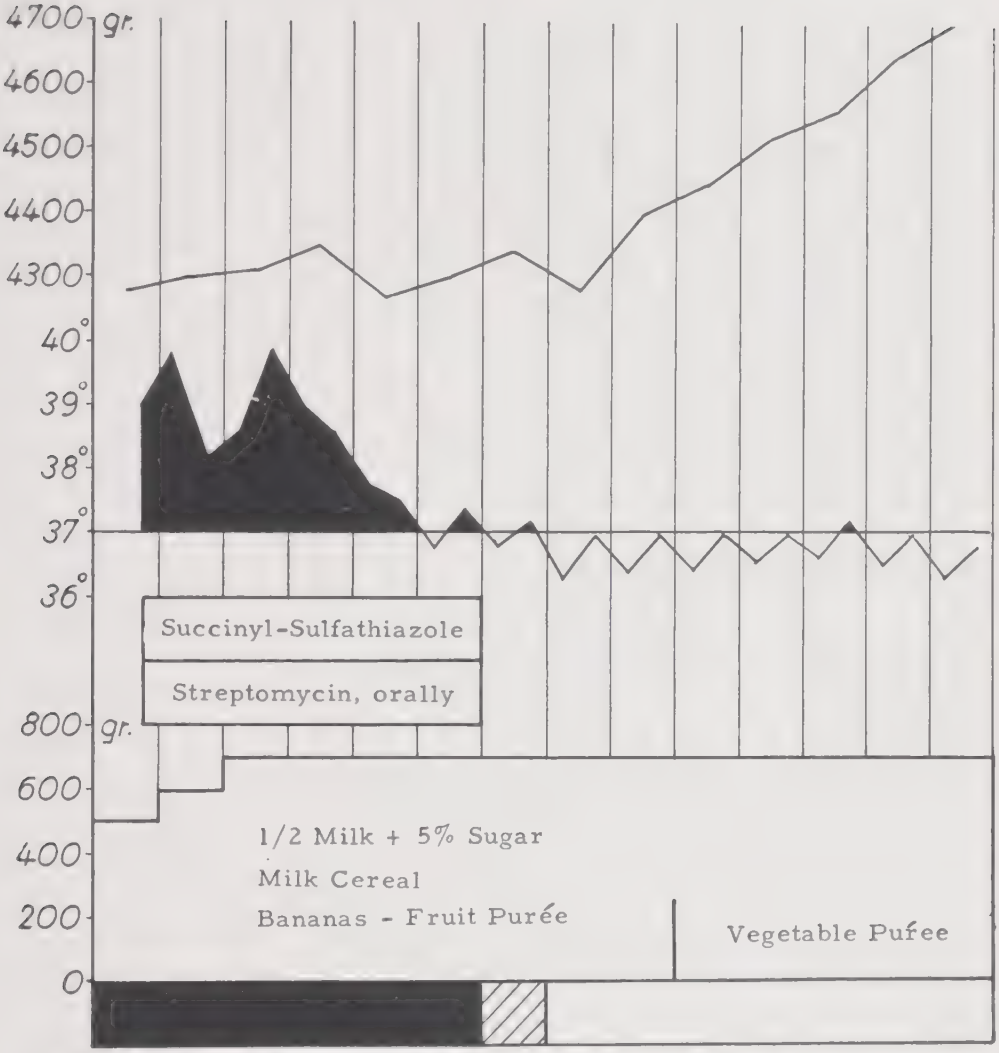


Fig. 58. Infant G.S., six months old. Dystrophy with signs of avitaminosis B and C. Stool culture: salmonella-enteritis Gärtner. High fever. Frequent at times bloody diarrheal stools but no toxic symptoms. Cure with almost normal diet. Medication: succinylsulfathiazol and streptomycin.

dietary therapy (12 to 24 hours only sugar water per mouth and possibly infusions), provided there are no other disturbances in the general condition. Feeding is continued with small quantities of a fatless diet given in frequent small meals.

Drug therapy of paratyphoid fever has made progress in recent years. In severe nervous complications, the encephalitic irritative symptoms or in encephalitis with its hyperpyretic temperatures treatment has, however, remained symptomatic. Protracted cool baths, regular doses of pyramidon or quinine in rather liberal quantities, and anti-convulsive drugs help at times to overcome this phase of the most severe forms of paratyphoid fever. Atropine, luminal and similar drugs show only little effect against vomiting. Interruption in oral food intake and parenteral feeding are the best methods to overcome this period of tendency to vomit which originates centrally. In case of deep somnolence tube feeding might become necessary when the child refuses to take food. No success can be expected from antibiotics in case of encephalitis.

The use of sulfa preparations, even in combination with streptomycin, has not brought any convincing success. On the other hand, chloromycetin, aureomycin and terramycin seem to be effective in salmonella infections. The effect on the general condition is at times good. The fever decreases after four to five days, although not in all cases. Relapses can, however, not be prevented even with chloromycetin, etc. They occur with the same frequency as in those patients where the fever dropped spontaneously, the diarrhea ceased and perhaps a better immunity developed. The number of patients who have become permanent carriers has rather increased with the use of antibiotics. The tendency to vomit makes the oral administration of the preparations, some of which have an unpleasant taste, more difficult. Rectal administration of aureomycin is ineffective since the preparation is not sufficiently absorbed and stronger diarrhea can make rectal treatment impossible.

Patients suffering from paratyphoid fever, with its erratic and unpredictable course, require constant observation so that sudden dangerous symptoms such as convulsions, collapse, etc., may be recognized in time and treated symptomatically. Paratyphoid osteomyelitis or paratyphoid pneumonia are serious illnesses. In such cases, too, the effective antibiotics should be given in adequate quantities. The prognosis of paratyphoid fever in infants must be made with caution. The unexpected development of severe complications and the transition into highly severe forms is always possible. Convalescence is slow. The dangers of recurrences and relapses or of secondary infections can be dismissed only after eight to 14 days of complete absence of fever.

The lack of appetite, which sometimes persists for a long time, delays final recovery. A small number of children remain carriers of bacteria for a longer or shorter period of time. Such children should then be kept in isolation since even antibiotics cannot alter this condition.

PARENTERAL INFECTIONS

The effects of infectious diseases of any kind upon the functions of the metabolism are at no time as pronounced as during infancy. At this age many infections manifest themselves not only in form of a local disturbance of one organ but, in contrast to the older child and to the adult, the digestive processes which are of such decisive significance for the development of the child are also affected. The harmful influence of infections cannot be proved in every child and in every case of illness. There is, however, always a possibility that such disturbances of the digestive processes might develop. The many possibilities in which an infection can affect the general condition and the reciprocal involvement of infections and nutritional disturbances frequently make it difficult to judge a pathological condition correctly.

Infections manifest themselves essentially in four forms:

1) The infection produces gastric symptoms: regurgitation, vomiting, lack of appetite.

2) The infection produces diarrhea or a diarrheal disease.

3) The infection produces a state of dysergia.

4) The infection produces a disturbance in the intermediary and absorptive metabolic processes.

All these disturbances which are caused by an infection lead sooner or later to a deterioration of the nutritional state and to a state of dystrophy.

GASTRIC DISTURBANCES

Loss of appetite is frequently the first sign of an incipient infection during infancy, which often precedes the onset of the disease by several days. The children appear pale during this time and the tongue is coated. Appearance of the coated tongue varies with the type of illness. Lack of appetite accompanies many febrile diseases during their entire duration. While some children still accept liquid food other patients refuse any type of food.

With difficulty it is possible to still give them small quantities of sweetened tea or cold water. The dryness of the oral mucosa resulting from the fever and the reduction of salivary secretion increases the difficulties in food intake. Many patients with fever prefer cool or cold liquids to the customary warm food. Some children take their food better when the liquid is acidified by the addition of fruit juice (lemon juice). Other children again refuse any strongly sweetened food. In such patients ordinary sugar can be replaced by glucose. The intake of fluids in sufficient quantities is important for all patients with elevated temperatures. The supply of sugar in some form reduces the protein consumption in febrile conditions. In somewhat older infants in a good nutritional state there should be no hesitation about discontinuing all other foods during the usually brief febrile illnesses. If the child is forced to take milk, cereal, etc., it frequently results in vomiting which is more harmful for the child than to forego normal nutrition for a few days, particularly because of the additional water losses. In prolonged febrile conditions the lack of appetite can become a serious problem. Today it is possible to overcome even prolonged periods of anorexia by parenteral feeding (water, glucose, salt, amino-acids) and by providing an adequate supply of vitamin B and C. At times an intravenous blood transfusion or, if hypoproteinemia has developed, an infusion of amino acid hydrolysates seems to improve the anorexia. There are no drugs which increase or produce appetite. At times children with prolonged periods of fever might accept food if the temperature can be reduced for a few hours by giving pyramidon or cool baths. Other children again will accept food if they are placed in a semi-sleep through sedatives.

Complications of the primary infection such as otitis or pyuria, diseases which have a particularly disturbing influence on the appetite, must be looked for. Unpleasant tasting antibiotics such as aureomycin or chloromycetin do not seriously disturb the appetite, at least in younger infants. Sulfa preparations, on the other hand, exert in some children an unfavorable influence upon the appetite.

The lack of appetite which follows a parenteral infection can be considerable and can persist for quite some time, without any apparent explanation. The secretory functions of the stomach are undisturbed. At times the anorexia might be caused by anemia or by a hypoproteinemia which remains as a result of the disease. Such conditions must be treated. Unpleasant memories of pains while swallowing, of a forced feeding connected with a feeling of suffocation, might at times contribute to the development of such conditions (see p. 525). No drugs are known which remove these difficulties of feeding following an infection. Hydrochloric acid, pepsin, etc. are ineffective. Adequate supply of vitamin B and C, if necessary by parenteral route, is important. Fresh air, lukewarm baths, possibly with addition of bath salts, followed by a massage of the body, ultraviolet irradiations, intravenous blood transfusions and similar procedures seem to help at times in the struggle against obstinate anorexia. Much depends on the skill and the imagination of the person caring for the child, who must find a way and a method to overcome the patient's refusal to accept food. For this reason it is at times of advantage in stubborn cases to place the child in a children's institution and to transfer the feeding to someone else. The mother who is nervous, impatient and discouraged by the constant failures frequently no longer possesses the calmness which is necessary for overcoming this difficulty. The last resort in stubborn, post-infectious anorexia is feeding by tube. In such a case any attempt to feed the child in the customary manner must be foregone for two to three weeks. The child must forget the unpleasant associations which were connected with feeding by nipple, spoon or bottle, in the customary mealtime situation. If it is possible to feed the child in this manner for some time with a diet which contains all necessary nutritive substances, and if the infant gains in weight for some time, a cautious attempt can be made after this period to return to the customary methods of feeding. As long as tube feeding is continued the food must contain all vitamins, egg yolk, possibly butter. It is important to provide adequate, even more than adequate quantities of protein in order to remove the hypoproteinemia which frequently causes the anorexia in the child. Difficulties in tube feeding arise when the child vomits the food. Luminal, given 20 minutes before the meal, feeding the child in a sitting position, or placing the child in a half-erect position following the feeding, can be tried in order to prevent the regurgitation of the food given by tube.

The degree of anorexia is of decisive significance for the speed with which a disturbance in growth develops. A complete refusal of food at any stage of a parenteral infection leads within a short time to severe damages, particularly in the young child. Lesser degrees of anorexia lead only gradually to a deterioration of the nutritional state. The insidious development of growth disturbances and the slight degree of anorexia are frequently more dangerous for the patient than a complete refusal of food which alarms those responsible for the child. The physician who sees the patient every day can easily overlook the progressing development of dystrophy. The seriousness of the situation is recognized only after the deterioration has progressed considerably.

Vomiting occurs almost always at the onset of an infection. Often it is the earliest sign of disease, preceding the rise in temperature. If a well developing breast fed infant suddenly vomits once or several times, it is correct in almost all cases to predict the onset of a febrile infection within the next 12 hours. Occasionally the child vomits only once. In other patients incessant or intractable vomiting develops. Single or repeated attacks of vomiting can, in infants with hydrolability (see hydrolabile children, p. 514) lead within a short time to weight losses of several hundred grams. If vomiting persists the children become pale and weak; their eyes become haloed. The patients are dehydrated and a metabolic alkalosis can develop. In order to stop this vomiting which introduces the infection, it is advisable to interrupt the food intake for some time. During this period the children receive every 10 to 15 minutes cold sweetened water to which a small quantity of salt is added in order to compensate for the chlorine losses in the vomitus. The tendency to vomit can be overcome with pyramidon given by mouth or as suppository, and by intramuscular injections of a barbiturate in adequate quantities. In the most severe and stubborn forms of this centrally originating vomiting it might be necessary to forego all food intake by mouth for 24 to 48 hours. The fluid and nutritional requirements of the children can temporarily be met by an enema administered by the drip method, of a 15% glucose solution to which one to two drops of tinctura opii are added. At times it is possible to influence the nausea by single gastric lavage. Repeated lavages and fillings of the stomach are not indicated in infants since they sometimes lead to atony of the stomach.

PARENTERAL DIARRHEA AND DIARRHEAL DISTURBANCES

Diarrhea is a symptom frequently accompanying febrile diseases in infants. This tendency to react to any infection, regardless of where it occurs, with frequent stools represents the essential difference between the earliest period of life and any other age period. In small children, school children, and adults the reaction of the intestinal tract to a parenteral infection manifests itself in almost all cases in form of a constipation. Changes in the neuro-vegetative influences upon intestinal functions probably cause these differences in the various age groups.

The effect of an infection upon the intestinal processes can attain any degree of severity, from a minor harmless diarrhea to severest acute diarrheal diseases. The minor effects of a parenteral infection upon the gastro-intestinal tract are much more frequent than the serious gastro-intestinal secondary symptoms of an infection. Even in the initially harmless but prolonged cases of diarrhea which might persist even beyond the period of the infection, vital substances are lost after some time so that an eutrophic condition cannot be maintained. The minor forms of involvement of the gastro-intestinal tract in case of infection are so frequent that in any attempt to find the cause of a diarrhea the presence of a parenteral infection must be considered primarily, particularly if the development of the infant was satisfactory until the time of the disturbance. It is not always easy to prove the presence of a parenteral infection in the beginning. Even before any symptoms of the infection become apparent vomiting and diarrhea occur. These pathological symptoms are then incorrectly traced back to some alimentary cause, including the so-called "upset stomach" which does not exist in infants.

Which symptoms permit a differentiation between diarrhea which accompanies a parenteral infection and a primary diarrheal disease?—In case of diarrhea caused by a parenteral infection the onset is sudden and surprising. Gastric symptoms, regurgitation and vomiting of unusual violence occur already at the beginning of an infectious diarrheal disease and continue throughout the disease, in fact they at times dominate the entire clinical picture. The anorexia, too, is usually particularly pronounced. In diarrheal disturbances occurring in the course of parenteral infections we frequently observe a discrepancy between the height of the fever, the amount of weight loss and the intensity of intestinal symptoms. High temperatures of about 39 to 40°, with a weight loss of only 50, 100 or 150g in the older infant, speak for a primary influence of a parenteral infection. Diarrhea, which develops only several days after the onset of high temperatures, is also characteristic for the parenteral infectious genesis of the disturbance. Finally an attempt will have to be made to confirm the infectious genesis of the disease by finding the parenteral focus of the disease (common cold, otitis media, bronchopneumonia, pyuria, etc.). This finding, however, can prove the infectious origin of the disturbance only if it is established during the first days of the disease. For even in primary diarrheal diseases many kinds of secondary infections occur not infrequently in the damaged organism.

The most essential differential diagnostic indications between a diarrheal disease caused by a parenteral infection and a primary gastro-enteritis (possibly caused by enteral infection) are as follows:

	Diarrheal Disease Caused by Parenteral Infection	*Primary Diarrheal Disease*
Onset of disease	Sudden, not preceded by dyspepsia	Frequently preceded by dyspepsia, aggravation of diarrhea
Temperature	High compared to the general symptoms	Slight, or high only in cases of severest general disturbances
Gastric Symptoms	Always present, frequently violent	Not marked
Reaction to Hunger Period	No or only partial drop in temperature	Obvious decrease of temperature, at times down to normal
Spontaneous Improvement	Possible	Possible only in light forms

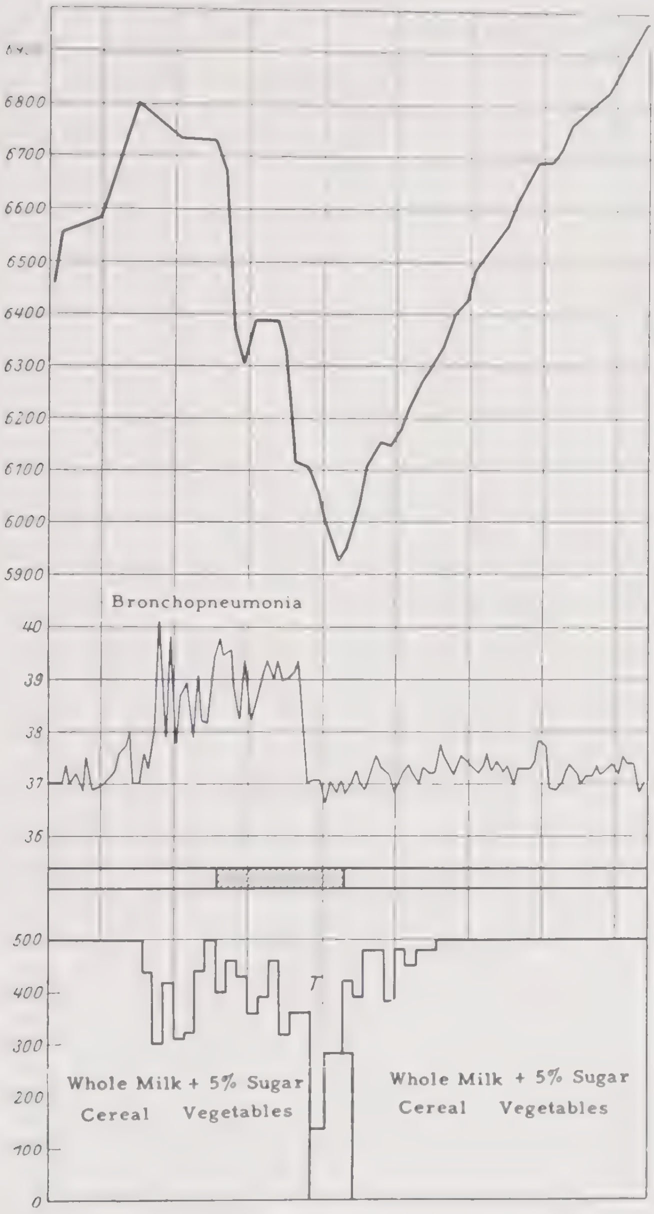


Fig. 59. A sudden drop in weight occurred around the eighth day of illness in the course of a clinically proved acute infection (bronchopneumonia). Poor food intake, repeated vomiting and fever could be given as an explanation since diarrhea to any severe degree is missing. The error was uncovered by the result of therapy; after temporary starvation and reduction in food the weight loss is stopped, vomiting ceases and the fever drops abruptly.

All these signs of diarrheal diseases caused by parenteral infections frequently do not suffice to recognize the nature of the disturbance on the basis of clinical symptoms. If the etiology of the disease is doubtful the therapy which is applied should always be that of a primary diarrheal disease. The course of the disease frequently brings clarification. Thus disturbances caused by infection can to a certain degree be excluded by the reaction to a hunger period. If an interruption in food intake fails to bring about improvements of the pathological symptoms this absence of improvement speaks rather for the presence of a parenteral infection. Occasionally, however, a fever caused by parenteral disturbances will also drop during the period in which only water or sweetened water is given to the patient. This influence of hunger upon a definitely confirmed parenteral infection is explained by the peculiarity of the water metabolism in case of infection (see Fig. 59).

On the basis of these experiences patients with high temperatures should not be forced to eat. Concentrated mixtures which usually are also rich in proteins are particularly unsuitable for infants suffering from an acute infection because of their considerable water requirements.

Diarrheas due to parenteral infections and primary diarrheal diseases differ also in their course. Primary gastro-enteritis has a tendency to become aggravated if it is not adequately treated. Diarrhea caused by parenteral infection, on the other hand, tends to improve spontaneously even without therapeutic intervention if the infection lasts for only a short time.

Not all infections exert the same harmful influence on the digestive processes. Primarily the so-called grippal infections, from a cold to a pneumonia, are with great regularity accompanied by diarrhea in infants. Diseases of the urinary passages react somewhat similarly. It happens not infrequently that a pyuria is overlooked in the beginning when it simulates the picture of an acute dyspepsia or a gastro-enteritis. Diseases of the skin and the bones have much less effect on the gastro-intestinal tract. Erysipelas, phlegmon, osteomyelitis, etc., are only rarely accompanied by diarrhea. Even severe diseases of this nature, accompanied by high temperatures, occur without disturbances of the intestinal activities, which shows that the changes in the gastro-intestinal functions are not caused by the height of the temperature and the severity of the disease itself.

The problem as to the connection between parenteral infections and intestinal diseases is difficult to answer. Is it a disturbance which originates centrally or does the intestinal tract suffer some kind of local damage? Does a disturbance of the peristalsis develop directly and thus result secondarily in changes in the digestive mechanism which are the basis for the development of an acute diarrheal disease, or are the secretion of digestive juices and the ability to regulate the bacterial flora primarily damaged by the infection and the fever so that the conditions for an acute diarrheal disease are created in this manner? Is it damage to the liver which was caused by the febrile infection and which resulted in acute disturbances in the metabolism? Several factors support the theory that a central-nervous disturbance (perhaps in the sense of "stress") produces the parenterally caused diarrheal disease. The special behaviour of the vegetative system in the infant might be of significance for this phenomenon. Only a minor increase in tonus might suffice in the vagotonic infant to bring about a diarrhea. The older child and the adult, whose vagotonus is lower, react to a parenteral infection not with diarrhea but with constipation, in contrast to the infant. But even this theory explains only the increase in peristalsis. In these cases, just as in acute diarrhea and gastro-enteritis, we lack any definite knowledge as to how the typical bacterial invasion of the small intestines develops.

Therapy of Parenteral-infectious Diarrhea

As long as the diarrhea which accompanies the infection persists as the sole pathological symptom it is best to merely observe the patient for a while. This applies particularly to the breast fed child in whom weight losses, diarrhea, vomiting and bad general appearance disappear within three to four days when the majority of infections heals. But even in the artificially fed child an initial period of observation is permitted when the physician has ascertained that the disease is caused by an infectious process. Such a period of watchful expectancy at times requires courage when the intestinal symptoms are violent or when larger weight losses accompany the disease as occurs not infrequently in hydrolabile children. The decision to forego any basic changes in the diet is, however, at times in the interest of the child. This is true particularly for severely dystrophic and atrophic children who frequently suffer from infectious diarrhea and in whom repeated hunger and therapeutic diets cause renewed damages to the child. In addition, infusion therapy might be dangerous in children with damages to the circulatory system, e.g., in the course of pneumonia, because of the additional demands on the circulation. Also in hydrolabile children who react to minor infections already with considerable weight losses, repeated hunger therapy leads within a short time to dystrophy and atrophy.

Definite symptoms of an acute diarrheal disease require changes in the diet. If there is any doubt as to whether the infection was the decisive cause in the development of the disturbance, a temporary complete replacement of the food by sugar water (10 to 20% dextrine-maltose solution, 500 to 700g per day) offers a possibility to determine to what extent the water metabolism is involved. Return of the temperature to normal or at least a drop by one to two degrees proves the existence of a state of water deficiency. If a drop in temperature takes place following interruption in food intake the food quantities can then gradually be increased. The detailed procedure in a five to six months old child should be as follows:

First day: Five hundred to 700g 15% dextrine maltose solution.

Second day: Reduction of the sugar solution to about 300g, given in two meals, plus two meals of a therapeutic feeding mixture such as 100 to 150g buttermilk with 5% sugar.

Third day: Further reduction of the sugar solution to about 100 to 200g, increase of the therapeutic feeding mixture to 300 to 400g per day.

Fourth day: Discontinuation of sugar water; 500 to 600g of the therapeutic feeding mixture, etc.

All cases of acute diarrheal diseases accompanying infections are treated by the same method as primary diarrheal diseases. However, difficulties occur not infrequently due to the severe gastric reactions of the child suffering from an infection. The necessary water supply by mouth fails because of the uncontrollable vomiting of the child. In such cases an attempt must be made to meet the water and food requirements of the child by parenteral fluid supply (see p. 394).

PARENTERAL INFECTIONS AND DYSERGIA

The treatment of dystrophic and atrophic patients who suffer from one or from repeated infections confronts the physician with serious problems of nutrition. At times it is possible to circumvent hunger on one hand and the damages caused by the food on the other hand by giving frequent, small meals. The possibility to control the bacterial flora in the intestines with antibiotics (streptomycin, chloromycetin, aureomycin) facilitates the situation. Parenteral feeding through infusions with plasma, blood and amigen permits a combination of a hunger therapy with an at least temporarily adequate food intake, particularly in children with severe vomiting.

Every infection is followed by a period of lowered resistance. The first infection which is accompanied by a lack of appetite, by diarrhea, by an "internal hunger" causes a state of starvation which leads to dystrophy. Dystrophy in turn leads to dysergia. These disturbances in well being, with simultaneous frequent infections, occur particularly in the mass care of institutions. They cannot be entirely avoided there even with careful nursing and nutrition. The successive infections in these cases increase with regard to their duration as well as their severity. Rapidly recurring infections, with constantly increasing duration and height of fever and corresponding shortening of the afebrile intervals are particularly dangerous. If the dysergia cannot be overcome the succession of fever attacks finally leads to a severe, possibly fatal disease.

In preventing and fighting dysergia an attempt must be made, provided the nutrition is adequate, to avoid as far as possible the development of new infection. A strict isolation is required particularly in young children who had already suffered from two or more infections in rapid succession. It is advisable that such children be isolated in institutions in closed cubicles with an experienced special nurse caring for the patient. Only if renewed recurrences can be avoided for a longer period of time is it possible to repair the damaged nutritional processes and restore the lowered immunity. The restoration of the lowered immunity is the more difficult part of this task. A number of these children will suffer again and again renewed attacks of "endogenous reinfections" despite strict isolation. Blood transfusions of 100 to 200g are effective in raising the immunity; they should be given intravenously in about weekly intervals. The medication of penicillin or sulfa preparations, continued over a prolonged period of time even after the temperatures have returned to normal, give the child the possibility to restore his cell functions and to rebuild his lost resistance. Irradiations with an ultraviolet light are ineffective for raising the immunity. In dysergic children it is essential to add vitamins, particularly B and C, to the patient's diet. Vitamin A is not an anti-infectious factor, as has been believed at times. An adequate protein intake is important since any prolonged infection produces a hypoproteinemia and the normalizing of the protein reserves, particularly the increase in globulin quantities, improves the immunity.

Parenteral Infections and Intermediary Disturbances

The consequences of an infection upon the entire metabolism have as yet not been clarified to a great extent. By examination of the entire metabolism changes could be observed which make it understandable why the weight gain is disturbed in case of infection. A rise in temperature produces changes in the metabolism of protein, amino acids and of salts. Infections with different etiology also seem to bring about different changes. It is furthermore known that an infection causes chemical and biological changes in the blood serum which can probably be assumed to be quite similar for the protein of the individual body cells. The possibility has been discussed of a direct damage to the cells through toxins or of an indirect damage through functional disturbances of vital organs (e.g., the liver) or of a lack of essential nutritive substances which are consumed in larger quantities or not produced during infections.

Only two facts can be established on the basis of clinical examination. After overcoming an infection the protein, carbohydrate and vitamin requirements are above normal. The same diet which prior to the infection brought about a satisfactory development of the child now fails to produce weight gains, even if the stools are normal and there are no gastric symptoms. By adding protein and by increasing the carbohydrates by 10% and more a weight gain can again be obtained. The reason for this increased carbohydrate requirement following an infection remains as yet unclarified. The infection shows a similar reaction upon the vitamin requirements. Larger quantities of fruit juices (50 to 100g) are necessary following an infection. It has not so far been determined whether during an infection the consumption of the vitamin reserves is increased or whether the vitamin production suffers damage due to the infection.

CHAPTER X

Constipation

CONSTIPATION AND PSEUDO-CONSTIPATION

A real constipation exists only when the eliminations of stool take place not only more rarely than normal but when the stools are also of abnormal consistency, solid and containing little liquid. Rare elimination of pasty stools of normal consistency is described as pseudo-constipation. The elimination of pasty stools in intervals of three, five to eight days, with the child feeling well and showing satisfactory development, occurs particularly in breast fed infants during the first to second month of life. These conditions which do not require treatment have been described in an earlier chapter (see p. 133). Real constipation which exists from the time of birth, where the child does not eliminate meconium or stool, originates from a malformation of the intestines (atresia, stenosis). This includes also partial atresia of the rectum or an incomplete atresia of the anus, where small quantities of a pasty or solid stool are eliminated from time to time. The distal end of the atresia can be felt by examination with the finger if the atresia is located near the anus. In partial atresia it is at times possible to enlarge the opening or perforate the thin dividing membrane. By inserting Hegar dilators or rubber bougies and increasing the diameter from day to day for eight to 10 days the enlargement can be kept open. In cases of complete atresia no meconium is eliminated. Roentgen examination after filling of the large intestines with barium should be carried out not only in sitting position but also with the head hanging down, in order to determine the length and the location of the atresia. The anal opening is marked with a small metal piece. With the child suspended by the legs, with the head hanging down, the distance between the intestinal section which still contains air and the anus gives an approximate indication regarding the length of the atresic section of the intestines. Treatment is surgical (see also p. 207). Tumors, teratomas, reduplication of the intestines, etc. which are already present at birth or which develop in the course of the first year of life can lead to constipation. The diagnosis can be made by palpation and roentgen examination. In any stenosis which is located higher up or in complete atresia the rectum contains no stool. In general the finding of an empty rectum with existing constipation speaks for the presence of an organic, real constipation.

Constipation in the infant develops much more frequently as a result of anal pathology. Fissures, bleeding rhagades and hemorrhoides occur already during infancy and become the cause for stool retention. These changes occur usually as a result of excessively solid stools or due to wrong nursing manipulations on the rectum, for instance incorrect insertion of the thermometer in the rectum, superfluous enemas, etc. This type of constipation frequently persists even after the damages on the anus have been repaired. A conditioned reflex causes the infant to hold back the stool, associating the process with fear of the pain which had occurred. Vulvitis in female infants or small rhagades on the external opening of the urethra in male infants also cause pain and lead to a retention not only of urine but also of stool. Treatment of the disturbance consists in the removal of the painful lesion on the anus. It is important that great care be taken to keep the anal area clean by regularly removing all remnants of stool with water or preferably with paraffin oil and cotton, applying a salve (2% dermatol salve, 2% Noviform salve, Balsam of Peru) and taking care that the stools are soft. In the treatment of rhagades the use of a silver nitrate stick or a solution of silver nitrate is not advisable. This procedure results in a hardening of the borders of the fissures, making healing more difficult. All attempts to obtain soft stools and to heal the fissures with the aid of enemas are harmful. In this manner further rhagades can develop in the spastic rectum, since the insertion of the enema tube is possible only by using some force. It is preferable to insert a glycerine suppository or to give one half to one or two teaspoons of paraffin oil in order to obtain a soft stool. The medication of other cathartics, particularly of castor oil, should always be avoided in infants. If continued over a longer period of time the child becomes accustomed to it and does not learn to eliminate the stools by his own efforts. In the young infant any drastic cathartic can result in an acute diarrheal disease. Surgical treatment of fissures is never required in infants.

Constipation develops in the infant also as a result of abnormal spasms of the anus for which no cause can be found, or as a result of a congenital narrowing of the anus. The difficulties which are encountered in the attempt to examine these patients rectally or to insert a rectal catheter into the anus permit the diagnosis. It is probable that some of these patients suffer from disturbances of innervation which can at times be improved by giving atropine, belladonna or papaverine.

Constipation caused by faulty diet occurs in young infants who receive a one-sided diet consisting of excessive quantities of milk without adequate addition of sugar. The constipation develops due to a carbohydrate deficiency (see chapter on nutritional disturbances, carbohydrate deficiency, p. 248). In older infants toward the end of the first year of life we occasionally observe a constipation although the children receive a normal diet corresponding to their age. The quantities of milk and protein which are contained in the diet and which are well tolerated by the majority of children and do not disturb the stool formation are apparently too great for these children with a predisposition to constipation. A stimulation sufficient to promote normal peristalsis is missing. By replacing the milk with malt coffee and sugar and increasing the quantities of meat or fish, vegetables, fruit and bread, and by adding jam or honey it is usually sufficient to cure this sluggishness of the intestines.

Constipation as symptom of hypothyroidism has already been mentioned (see p. 237). The medication of thyroidin in adequate amounts cures this form of constipation.

Patients with severe cases of active rickets also frequently suffer from constipation. Hypotony of the musculature, meteorism and disturbances in the vegetative system cause this constipation which at times continues far beyond the acute stage of rickets.

MORBUS HIRSCHSPRUNG AND CONGENITAL MEGACOLON

Constipation is the leading symptom in Hirschsprung's disease and in congenital megacolon, diseases which not infrequently develop during infancy. Megacolon and Hirschsprung's disease should be differentiated today since the anatomical changes are different in the two diseases. Accordingly, the therapeutic procedure in congenital megacolon is different from that applied in Hirschsprung's disease.

In congenital megacolon there is probably a congenital, functional enlargement of smaller or larger parts of the sigmoid and the colon, which continues to the rectum and the anus. In making the diagnosis of megacolon it must be remembered during sigmoidoscopy that a long, loose and twisted sigmoid is a physiological phenomenon in the infant. The significance of valve formations and similar changes or of kinking as cause of a megacolon or megasigmoid has been grossly overestimated. During sigmoidoscopy the intestines with their thin walls can, in the young child, be dilated so considerably by filling that they occasionally simulate the picture of a megacolon. Good pictures are obtained if the filling of the lowest parts of the intestines is accomplished from above. The roentgenological demonstration of the large intestines following a gastro-intestinal passage of barium gives a more correct picture concerning their condition than sigmoidoscopy.

In Hirschsprung's disease a dilatation of the sigmoid and the colon develops secondarily as a result of an underdevelopment or absence of the plexus myentericus in one section of the rectum. The cells of the ganglia of Meissner's and Auerbach's plexus are partially or totally missing. The plexus is absent in the lower rectum in 100% of the patients, in 60% of these patients also in the area of transition from rectum to sigmoid, and in 20% also in the sigmoid. In very rare cases defects of the plexus myentericus have been observed throughout the entire large intestines and even as far as the small intestines. The section of the intestinal tract which is not or incorrectly supplied with nerves is, as a result of this deficient supply of nerves, narrowed to the thickness of a pencil. The feces accumulate in the section of the intestines above the narrowing. The intestines are gradually dilated and distended and the intestinal wall gradually becomes hypertrophic.

Hirschsprung's disease exists or develops already during early infancy. It occurs occasionally in siblings or in several members of a family. In the individual cases within a family the form of the disease varies from the picture of a stubborn constipation to severe extended intestinal atresia.

The first symptoms of megacolon can develop already shortly after birth. The child suffers from meteorism and constipation which is occasionally accompanied by vomiting. The distension of the intestinal tract increases in the course of the following months gradually but constantly. Signs of obstruction of the intestinal passage are, however, absent. There is no visible peristalsis. In the roentgenological examination large masses of gas are found in the intestines but no fluid levels can be observed.

Rectal examination permits to a certain degree a differentiation between congenital megacolon whereby the intestines are dilated up to the anus, and Hirschsprung's disease, where a longer or shorter narrow piece of intestine is found between the anus and the dilated intestinal tract. In case of megacolon rectal examination reveals a filling of the intestines with feces up to the sphincter. In Hirschsprung's disease the palpating finger reaches a relatively narrow section of the intestines which is empty. In megacolon a mucous secretion flows continuously from the anus and irritates the area of the anus. In Hirschsprung's disease the area around the anus is dry.

The symptoms of Hirschsprung's disease usually do not manifest themselves until the second half year of life. Here too meteorism develops. In the large intestines large quantities of thickened feces can be palpated. This symptom can, however, at first be absent during infancy. Visible peristalsis is not infrequent. The children appear grey. They are in a bad humor. Food intake becomes more and more difficult because the distended intestines trouble the child. Large masses of pasty or solid stools are eliminated only in intervals of several days, or a stool elimination can be effected only with the help of enemas.

In congenital megacolon the treatment with acethyl cholin per mouth or by injection has been found effective in removing the disturbance of balance in the vegetative system. The children receive after the first meal acethyl cholin (0.05 to 0.1g) or dihydroergotamine (three times daily five to 20 drops), and in the evening one to two teaspoons paraffin oil. If no results are obtained with these medications a second dose of cholin is given in the afternoon. If this treatment is continued for one to two months, a normal elimination of the intestinal tract can be brought about. According to the roentgenogram the dilatation of the intestines persists, but the mechanism of elimination becomes normalized. During the first days of treatment the intestines should be emptied regularly with the aid of enemas.

Treatment of Hirschsprung's disease consists of surgical removal of the spastically contracted section of the intestines, including a piece above the narrowed section and the rectum down to the anus, with conservation of the anus. This extensive intervention is necessary since the anatomical changes extend in almost all cases beyond the visibly narrowed section. The resection of the rectum in Hirschsprung's disease, which if possible should be delayed until the second year of life, has today become relatively safe due to the preparatory treatment of the patient with streptomycin and slowly absorbing sulfa preparations which sterilize the large intestines. The attempted drug therapy in Hirschsprung's disease and the severance of sympathetic fibers through resection of the corresponding sections of the plexus has at present been abandoned since the narrowed section of the intestines which only secondarily caused the dilatation of the adjoining proximal intestinal section remains intact and after initial apparent improvement relapses are to be expected.

CHAPTER XI

Nutritional Disturbances of the Breast Fed Infant

The milk which the infant sucks from the breast is always of suitable quality, is never harmful in its composition and is never spoiled. Laboratory examination of breast milk is never necessary. The results could never be used to explain a disturbance in the development of the breast fed infant. Assuming that the diet of the mother is adequate breast milk contains all nutritive substances which are necessary for the normal development of the child, at least for the first four to five months of life. Thus the large number of nutritional disturbances which for the artificially fed infant are a frequent source of disease and developmental disturbances, are completely eliminated in the breast fed infant. For the breast fed child which drinks sufficient quantities scurvy, insufficient intake of carbohydrate and, at least up to the seventh month of life, protein deficiency do not exist. Only rickets makes an exception. It occurs in the breast fed child as frequently as in the bottle fed infant, although it usually appears in the breast fed child in a lighter form. The amount of antirachitic vitamin D contained in breast milk is not adequate for either prevention or healing of rickets. The perfect quality of breast milk, which is never spoiled, furthermore results in an absence of acute diarrheal diseases in breast fed infants, while in artificially fed infants they develop as a result of spoiled milk. This eliminates almost all severe forms of acute diarrheal diseases as well as toxic gastro-enteritis, which develop as a result of incorrectly composed or spoiled food.

Nutritional disturbances in the breast fed child develop rarely from an excessive milk intake, more frequently from an inadequate intake and from enteral or parenteral infections. A peculiar constitutional quality of the tissues of the infant also manifests itself in the breast fed child. In the breast fed infant, therefore, the etiology of a nutritional disturbance is limited and easily recognized, in contrast to the many possibilities in the artificially fed infant. The rare occurrence of acute nutritional disturbances also offers an explanation for the fact that in former years, when the methods of artificial nutrition were far from adequate, morbidity and mortality was far lower in breast fed infants, even under unfavorable external conditions, than in the artificially fed infant.

Disturbances in the development of breast fed infants can appear only from the following causes:

a) From an incorrect quantity of food, which can be either excessive or insufficient.

b) From infections, particularly parenteral non-specific infections or more rarely from specific intestinal infections.

c) As a result of a special quality of the tissues. Constitutional disturbances.

OVERFEEDING AND UNDERFEEDING OF THE BREAST FED INFANT

In former years overfeeding played an important role in the pathogenesis of nutritional disturbances in breast fed infants. A number of physicians still believe today that disease and lack of development can frequently be caused by an excessive food intake. Overfeeding with breast milk is, however, only rarely the cause of a disease. It occurs occasionally in premature and weak children who are placed on an already abundantly flowing breast. But even in these cases the nutritional requirements of the child and the output of the breast adjust to each other within a short time so that the excessive food intake is soon regulated. In the full term child overfeeding is also avoided by regurgitation of the excess of milk. Furthermore the tolerance for natural food, even in quantities which exceed the demand considerably, is so great in the healthy child that excessive intake does not cause disease. The results of temporary overfeeding are increased fat deposits. In this manner the child can be fattened considerably, a condition which is undesirable but which hardly ever causes a disturbance. Weak and premature children who receive their food through a tube or in a bottle can be harmed by inadequate dosage of their food intake. If overfeeding causes a disturbance it develops in form of an acute diarrhea. Regurgitation and vomiting, diarrhea, absence of weight gain or even weight losses occur.

Treatment of overfeeding is simple. Food quantities must be reduced. This applies to full term children as well as to premature and weak infants. An intake of 100 calories per kilogram of body weight should initially be considered sufficient. This reduction in food intake can best be accomplished by shortening the drinking period to about 5 minutes. In other cases it might be advisable to express part of the milk manually before placing the child on the breast. While in cases of overfeeding the number of meals will remain at five during the third to tenth week of life, the number of meals can be reduced to four after this age.

Insufficient food intake in the breast fed infant is of far more practical significance than the rarely occurring overfeeding. While the importance of overfeeding used to be overestimated, the problem of insufficient food intake did not receive enough attention. The dangers of this insufficiency are frequently not correctly evaluated because the symptoms which develop in the breast fed infant as a result of hunger do not always point to the existence of a state of hunger. In all cases of poor development of a breast fed child, but also in cases of diarrhea and poor skin turgor as well as in cases of regurgitation and vomiting in a breast fed infant an insufficient food intake must be considered. By weighing the meals throughout one day—the determination of the weight at one or two meals is not enough—hunger can be either diagnosed or excluded as cause for the lack of weight gain.

This requirement is of particular importance during the first period of life. During the first eight to 14 days a relative hunger is a physiological condition which ceases only when the breast begins to secrete adequate quantities of milk or when the child has learned to ingest the quantities of milk which are supplied to him. This symbiosis between mother and child at times fails to develop from the beginning so that a relative insufficiency of milk intake beyond the physiological limit of the first two to three weeks of life occurs not infrequently. The absence of weight gain or a weight loss, also a delay in regaining the physiological weight loss after birth must be recognized in time and interpreted correctly. It is important that after the third week of life at the latest the child receives quantitatively sufficient food and shows a gain in weight. Any prolonged hunger is just as dangerous for the breast fed infant as it is for the bottle fed child. A state of dystrophy can develop in the breast fed child with the same disturbances which are characteristic for the dystrophies of bottle fed children. This includes primarily the loss of immunity and of resistance. The deterioration of the nutritional state develops in the breast fed infant more gradually than in the artificially fed child because the accompanying diarrheas are usually absent. In the long run, however, hunger damages the structure and functions of cells also in breast fed infants.

The majority of children develop, apart from the primary symptoms of insufficient or totally absent weight gain, constipation which can reach a stage whereby only rarely small amounts of dark hunger stools are eliminated. The abdominal wall of the children is sunken, the skin becomes withered and fails to show the beautiful rosy shimmer which distinguishes the skin of the well developing breast fed infant. These changes of the skin take place particularly when the hunger does not occur in later months following a period of adequate nutrition, but rather when it follows immediately the short physiological hunger period of the newborn. While an adequately fed infant loses the normal withered appearance of the first weeks of life the breast fed infant who suffers from hunger from the time of birth remains withered and becomes pale and grey. The children are restless and discontent. Their frequent crying is not always interpreted as an expression of hunger but at times as sign of overfeeding or of an unsuitable composition of the milk. Some mothers, led by these wrong conclusions, interrupt milk feedings because they do not seem to agree with the child, or they replace some of the meals by cereal water or strongly diluted milk mixtures.

Hungry breast fed infants are, however, not always restless and crying a great deal. Some of the children appear calm and satisfied, sleepy and scarcely moving. One obtains the impression that the child is not hungry. The hunger condition is in some children concealed by the fact that no constipation develops.

Diarrhea, accompanied by regurgitation and vomiting occurs ("inanition diarrhea," see p. 417). The hungry intestinal cell loses the ability to control the intestinal bacterial flora. This offers the possibility for the development of a state of irritation in the large intestines. In the breast fed infant, just as in the bottle fed child, there are types of diarrhea which can be cured only by adequate nutrition. In the hungry breast fed child the knowledge of the existence of "hunger diarrhea" is of importance because the symptom of diarrhea frequently leads to the erroneous assumption of overfeeding. If the milk quantities of a number of meals are weighed and found to be considerably below the quantum which is necessary to meet the caloric requirements of the infant, the diagnosis of underfeeding is certain. The second part of the diagnosis, which is the determination as to whether the cause for the insufficient food intake is to be found in the mother or in the child is at times more difficult. Troubles in drinking which were already mentioned and discussed in an earlier chapter and which develop as a result of laziness in drinking or of awkwardness of the child (see p. 124) can be determined by observing the child while sucking on the breast. Any one of these causes can lead to insufficient milk production due to the child's fault, even if the mother's breast initially secretes sufficient quantities of milk.

Primary lack of milk supply causes a state of hunger in the infant when the breast of the mother of a well and expertly drinking child fails to secrete adequate quantities of milk. This includes women who never achieve adequate milk secretion but who desire by all means to nurse their children and who are afraid of giving any additional foods. It also includes mothers whose breast milk secretion is initially adequate, frequently even seemingly abundant so that even during the intervals between feedings milk is constantly secreted from the breast. Particularly in these women we observe frequently already in the first month, at times only in the second or third month a reduction in milk secretion. These mothers, relying on their initially abundant secretion, continue to nurse their infants without giving additional food, and in this manner a state of undernutrition develops. In a third group of women the milk production does not diminish until the sixth to seventh month when it decreases rapidly so that even then an insufficient food supply is possible if the addition of other foods is delayed.

In treating the insufficient milk intake in breast fed infants an attempt must be made as far as possible to maintain the supply of natural food. If the difficulties in drinking lie in the child this aim can be reached relatively easily. The nutritional state of the child should be improved by the addition of other foods so that gradually the sucking weakness or, with increasing age, the awkwardness and laziness in sucking can be overcome. Placing the child on the breast at more frequent intervals is rarely successful. It is preferable to meet the nutritional requirements of the child by temporarily supplementing the diet. For such supplementation the following milk forms are suitable: expressed breast milk or, if this is not available, high caloric feeding mixtures such as whole milk with 10% sugar, slightly diluted sweetened condensed milk, farina prepared with whole milk and sugar—the latter food being particularly indicated in children where regurgitation and vomiting aggravate the state of hunger. Usually small quantities of these mixtures suffice to make up the deficiency in breast milk. Amounts of 50 to 200g daily of one of these mixtures, depending on the amount of food deficit, distributed among some or all of the meals, are usually adequate. It is advisable to give this food with a teaspoon, even to the youngest infants, so that the child will not become accustomed to the bottle which can be emptied more easily than the breast. With the exception of cereal, which is given before the meal in order to prevent regurgitation and vomiting, all supplementary food should be given following the breast feeding so as not to disturb the first hunger of the infant and the complete emptying of the breast. If after some time an improvement in appetite, in sucking and thus an increase in milk secretion can be observed, the supplementary food can be omitted and exclusive feeding on the breast can be continued. It is by far more difficult to treat a primary lack of milk supply which originates from a functional weakness of the breast glands. Despite numerous recommendations there is no effective galactagogue. An increase in milk secretion can better be accomplished by complete emptying of the breast which can also be effected with the help of breast pumps. However, it is advisable to avoid exaggerated activity in this direction so that the nursing mother will not suffer too great a nervous strain. Here, too, the addition of artificial food is preferable. At times it might be advisable to place the child on both breasts for only three meals a day and to give only artificial food for the other two meals. This transition to a mixed diet can be carried out particularly in older breast fed infants whose age already justifies the introduction of a cereal or vegetable meal. In no case, however, must a diarrhea of the hungry child lead to the introduction of hunger therapy or a therapeutic diet in the hope that the disturbing diarrhea can be cured in this manner. The frequent stools will become normal in the hungry breast fed child only when the nutritional requirements are fully met.

DISTURBANCES DUE TO INFECTIONS

Enteral or parenteral infections lead to diarrhea and diarrheal diseases—in breast fed infants no less than in bottle fed infants. Infectious diarrhea of the breast fed infant interrupts the hitherto undisturbed development of the child. The children have temperature elevations depending on the severity of the infection. The fever is frequently high compared to the relatively small weight losses and the other disturbances in the general condition. The discrepancy between height of temperature and minor disturbances of the general condition points to the fact that the disease is not a primary diarrhea or gastro-enteritis. The children are restless, they cry much and their sleep and appetite are disturbed. Vomiting, which is never absent and frequently very violent and which precedes the disease and persists throughout the period of infection speaks for the infectious genesis of the diarrheal disease. If vomiting sets in suddenly in a hitherto well developing breast fed infant it is the first symptom pointing to the onset of an infection. This symptom is of diagnostic significance in those children where the infection cannot be recognized without difficulty by high temperatures or by definite clinical symptoms but where the infection manifests itself in form of a slight cold or a pharyngitis. In such patients it is at times possible to determine the infectious nature of the diarrhea with the help of such hints as the appearance of more or less severe glandular swellings at the angle of the mandible or behind the sternocleidomastoid muscle or the presence of catarrhal diseases in the child's environment. A urine examination should never be omitted since also pyuria can begin under the camouflage of febrile diarrhea.

In any acute diarrheal disease in a breast fed infant the possibility of dysentery or paratyphoid fever should also be considered. These rather rare infections occur in breast fed infants frequently without the typical symptoms of mucous, bloody stools etc., but rather in form of a harmless acute gastro-intestinal catarrh.

The attempt to explain the connection between infection and gastro-intestinal disturbance meets in breast fed infants with the same difficulties as were encountered with regard to bottle fed children. Diarrhea is the customary reaction of the infant to any parenteral infectious damage (see p. 459).

In treating infectious diarrheas in breast fed children any radical dietetic measures should be avoided. If the infectious etiology of the diarrhea is recognized the physician should not overestimate the significance of the abnormal intestinal eliminations and of the vomiting. An attempt must be made to maintain in the nursing mother the confidence for continued breast feeding. The assumption that an inadequate quality of the milk, overfeeding or similar causes brought about the disturbance must be excluded since such conjectures lead to radical changes in the diet which then result erroneously in a discontinuation of breast feeding. Treatment of these disturbances is accomplished essentially by the child himself. The fact that the child has difficulties in breathing through the nose as well as the vomiting lead to a reduction in the amounts of milk taken by the child so that the child himself reduces his food intake. However, this reduced food intake is not without danger if the water requirements, which are increased during the period of fever, are then no longer covered. It is therefore an important task in the treatment to take care that the fluid requirements are met, either by giving tea sweetened with saccharine by spoon between the meals or by giving the child an infusion. Since severe vomiting is always accompanied by larger water losses it will always be necessary in children in whom

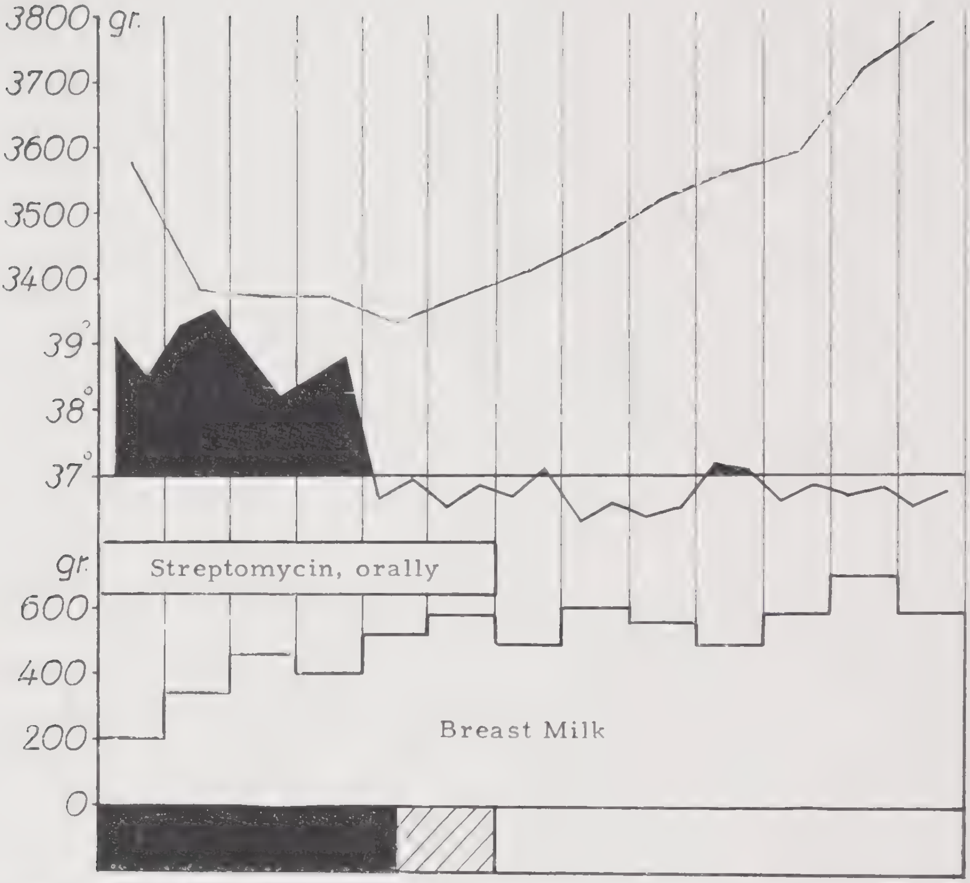


Fig. 60. Infant M.M., one and one-half months old. Grippal infection. Acute diarrhea. Cure without change in diet (breast milk). Streptomycin per os.

vomiting dominates the picture of the disease to calm the vomiting center by giving sedatives (luminal etc.). It is, however, wrong to introduce cereals in these cases of infectious vomiting since the intake of concentrated food increases the water requirements of the tissues.

Since uncomplicated infections are cured after an average of four to five days a return to normal stools can be expected after this time. This expected spontaneous recovery from diarrhea makes any radical therapeutic changes superfluous. Cessation of diarrhea occurs when the infection disappears.

In the large majority of infectious diarrheas a period of watchful expectancy will be sufficient. Only in cases of severe involvement of the nutritional processes, of severe weight losses and definite involvement of the general condition a special dietetic treatment will be unavoidable, just as in the bottle fed child: hunger period (tea), then starting with small quantities of 100 to 200g of food per day, with gradual increase. These more serious diarrheal diseases develop only rarely in breast fed infants. Very occasionally they occur in children where a transition to breast milk was made because of a nutritional disturbance due to artificial feeding. In such cases the diarrhea occasionally deteriorates into a toxic gastro-enteritis. The treatment is then the same as in acute gastro-enteritis of artificially fed infants (see p. 391). Toxic gastro-enteritis in breast fed children who hitherto had shown satisfactory development is almost always the result of a specific intestinal infection.

The prognosis of infectious diarrhea and diarrheal diseases in the breast fed child is good particularly because the diseases occur almost always in eutrophic infants who not only are able to overcome the infection rapidly but who can also recover rapidly from disturbances of the gastro-intestinal processes. The only exceptions are those children who were already damaged for some reason so that the reduced nutritional state somewhat influences the prognosis.

CONSTITUTIONAL DISTURBANCES IN DEVELOPMENT

In a very small group of breast fed infants no satisfactory development can be obtained with the customary feeding methods, despite the fact that all the basic requirements for normal development on the part of mother and child are present. This state of retarded body development is frequently accompanied by diarrhea. The children differ in their appearance from the ideal type of breast fed infant. The physiologically withered appearance of the first three to four weeks of life persists. The children resemble artificially fed dystrophic infants. They lack the rosy color of the skin, the skin appearing rather greyish. Regurgitation and vomiting occur. The children are restless and capricious so that even the quantities of milk taken at the individual meals show variations. As in the dystrophic child symptoms appear which point to a deficiency of individual organ systems. Skin and nervous system are primarily affected. The tendency to intertrigo is great. Scaly dermatitis develops in the face and on the body. These skin disturbances can appear in different degrees of severity, from localized scale formation and simple intertrigo to severe seborrheic dermatitis. Every day of continued dystrophy causes a deterioration of the pathological skin disturbances. The defects of the nervous system manifest themselves in form of symptoms which characterize the neuropathic infant. Restlessness, superficial sleep, itching of the skin impede the development of the child.

The abnormal condition of skin and nervous system improves if the attempt to obtain a weight gain is successful. The stool eliminations become normal even without special treatment once the dystrophic condition is removed. The nature of these dysplasias, however, remains unexplained. These dysplastic conditions are unusual because the pathological nutritional state develops despite a diet which, as far as we know, is quantitatively and qualitatively complete. The restlessness which characterizes many of these children results in an increased caloric consumption. Furthermore, in some of these children so much food is lost through regurgitation and vomiting that the absence of weight gain can be explained by these losses. In other children the same disturbance of the nutritional state can take place without pathological symptoms on the part of the gastro-intestinal tract. Diarrhea may perhaps lead to losses of mineral substances which become particularly obvious in children fed with the salt-poor breast milk which barely meets the requirements of the organism. It is not known to which extent these children suffer from disturbances of the assimilatory processes in the cells beyond the intestinal tract.

With all the doubts concerning the pathogenesis of these disturbances one fact is certain: the dysplastic condition can be remedied by adding protein and salt containing food mixtures to the diet.

The following mixtures are suitable for complementing the diet: buttermilk with 5% sugar, protein milk with 5% sugar or sweetened condensed milk or whole milk with 5 to 10% sugar. Frequently only the addition of a protein preparation (e.g., Casec) is sufficient. These milk mixtures are given in quantities of 10 to 20g, added to every meal. If this change in diet is not sufficient the amount of additional food must be gradually increased. In order to prevent overfeeding on an abundantly flowing breast it is advisable in some patients to shorten the nursing period on the breast to five to seven minutes or, instead of adding the feeding mixture with every meal, to replace one or two entire meals by one of the artificial feeding mixtures, in quantities which correspond to the age and the weight of the child.

One of the above methods will in almost all cases bring about an immediate rise in the hitherto horizontal weight curve. Diarrhea which might persist for another few days in undiminished severity improves after about four to eight days. Once the stool has become approximately normal the condition of the skin also shows improvement. Intertrigo disappears as well as the eczema-like and desquamative changes on trunk and face. The duration of the entire disease extends over a period of about two to three weeks.

The supplementary food compensates for a deficiency in an H-factor, the Biotin (see p. 266) which in breast milk is at times present in only very small quantities. It is possible that a normalization of abnormal intestinal processes due to the high protein diet also plays a role in the curing of the disease. As a result of the increased intake of amino-acids and of salts the requirements for these substances in these children are met.

The improvement of this state of poor development due to addition of cow's milk mixtures or protein preparations does not permit the conclusion that it is necessary to wean the child from the breast. On the other hand it is wrong to try to force a recovery with exclusive breast feeding.

This apparently simple pattern of treatment is in practice not infrequently met by certain difficulties. Nervous children often vomit violently and any additional food intake leads to an aggravation of the gastric symptoms. In such children the replacement of one or two breast milk meals by cereals is preferable to the addition of milk mixtures. The necessary complementation of food is at times rendered more difficult by the lack of appetite in these children. Small quantities of concentrated feeding mixtures (condensed milk, whole milk with 17% sugar, concentrated protein milk with 20% sugar) are necessary in these poorly drinking children in order to obtain an adequate caloric intake and a rise in the weight curve. Dystrophy accompanied by diarrhea can be cured in breast fed infants only if a weight increase can be accomplished, and for this purpose any means are permitted. Tube feeding might become necessary if complementation of the diet by other means is unsuccessful. With these therapeutic procedures the lack of development in breast fed infants, which in former years was frequently and erroneously described as constitutional hypotrophy and hypoplasia, has now become rare.

CHAPTER XII

Constitution and Nutrition

DAILY OBSERVATION shows that the development of one infant never resembles completely that of another. This fact becomes even more impressive when the same disease, for instance a nutritional disturbance, attacks different children. We can never find a disease which in all its symptoms is identical in different children. The basis of this difference in behaviour toward the same stimuli is today explained by a difference in constitution. As constitution we describe the sum total of the physical characteristics which the child possesses at the moment of birth and which to the largest extent are inherited from his ancestors. The constitution together with the characteristics which are acquired in the course of life through external influences produce the physical condition of the child. A disease is a momentary picture of the mode of reaction of a child at a certain time of his life.

The difference in constitution manifests itself in the healthy and sick infant more clearly than in the adult. It is to the merit of pediatrics (*Czerny*) to have emphasized the significance of constitution at a time when the origin and the form of disease were declared to be essentially due to the influence of exogenous causes. Today the influences of the child's constitution are amply discussed in every chapter dealing with the physiology and pathology of infants. In this book, too, the significance of constitution and physical condition with regard to the course of a disease has been stressed repeatedly. Constitution in this sense is a rather loose term. Any symptom and any expression of life of a healthy and a sick child is determined by the constitution and the physical condition of the child. The morphological and functional bases of the different modes of reaction of a child are not known.

For many years much effort and labor has been spent in recognizing the different constitutions on the basis of the habitus of a person. The Hippocratic division of human beings on the basis of the composition and mixture of their humor into phlegmatic, choleric, melancholic and sanguinic people was the first attempt which was undertaken in this direction, and was later followed by many others. In pediatrics, too, numerous attempts have been made to find foundations for a morphology of the constitution on the basis of the descriptions and systems of *Sigaud* and *Kretschmer*. *Sigaud* and his disciples *Caillou* and *McAuliffe* differentiate among four types: respiratory, digestive, muscular and cerebral. The muscular type corresponds to the athletic habitus, the digestive type to the pyknic type of *Kretschmer*'s classifications, while the respiratory and cerebral types are combined, according to *Kretschmer*, in the asthenic type. For the age of infancy *Sigaud*'s classification appears suitable.

The *typus muscularis* includes the well developing strong infants, with strong musculature and rosy appearance. Their extremities are rather short, the skull round, mouth, ears and nose small. Hair growth is sparse, the forehead low. In the *typus digestivus* all the parts of the body which are connected with digestion are particularly developed. The abdomen is large and protruding. They have short skulls. Hair growth is abundant, the hair borders curved into the forehead, the forehead low, the nose small, frequently turned up, and the jaws are large and strong. These children usually make no difficulties in their development. They are good eaters and feeding difficulties are rare. The *typus respiratorious* is characterized already during infancy. In the pure forms the thorax is long and narrow, the epigastric angle is small. The nose appears already at birth unusually large. The infants are skinny. There is no tendency to adiposity. From among them develop many of the so-called constitutionally skinny and weak children. The children belonging to the *typus cerebralis* are from the beginning small and thin, the chest is flat, the abdomen convulsions and for persistent vomiting with acetonemia. Diffi-facial contour narrows considerably toward the chin. Hair growth often is strikingly abundant, the hair frequently curled and thin. The border of hair growth protrudes at a sharp angle on the forehead or it appears concave, almost at the height of the skull. The ears are usually large, thin and standing off. These children usually present difficulties in their development. Neuropathic and psychopathic patients develop from this group. Vomiting and pylorospastic children also belong to the group. Sleep disturbances frequently occur in these children, and in later years they easily develop attacks of fear. Badly eating, stubborn children and others suffering from a number of nervous disturbances are observed rather frequently among the typus cerebralis. The development of an acute disease causes numerous difficulties in these patients. These are the candidates for the so-called febrile convulsions and for persistent vomiting with acetonemia. Difficulties in the care, in the taking of medicines and violent resistance against injections etc. occur frequently.

The influence of the constitution upon onset and course of the different forms of nutritional disturbances was overestimated in former years. Until not so long ago it was believed that the development of dystrophy or atrophy was in all cases due to an abnormal constitution of the child. These assumptions are to a certain degree justified. However, it is only a small number of children who react to faulty nutrition in this particular manner. It is erroneous to draw from this fact the conclusion that the endogenous factors in the child exert the decisive or even the sole influence in the development of an abnormal nutritional state. It is not permissible to explain the lack of development of a child with his constitution. Dystrophy or atrophy never develop without external disturbances. With the progress in the recognition of the causes of nutritional disturbances and in their treatment the number of so-called constitutional diseases diminished more and more.

A further group of children can be differentiated with regard to their reaction to food intake; these children will show a satisfactory development on the basis of their constitution regardless of their diet, regardless of whether too much or too little is given in comparison to the "norm," whether they receive large or small quantities of sugar and whether their vitamin requirements are met by external supply or not. Apart from these few fortunate children who show the same satisfactory development with unsweetened whole milk and without vitamin intake as with any other incomplete feeding mixture, there is a much larger group of infants who can develop satisfactorily only if the composition and the quantity of food meets certain requirements. This includes for instance an adequate intake of carbohydrates. On the other hand it can also be observed that growing children receiving the same diet show entirely different reactions with regard to their development. There are children who deposit fat easily and others who do not deposit fat at all. The same kind of nutrition can produce short or long children, etc.

The concept of constitution overlaps in some respects with that of individuality or personality. Constitution means a special quality of tissues and cells which is inherited and which in its reactions often recurs within families. There are, however, a number of infants whose cells and tissues show diseases and pathological symptoms even under normal living conditions, with normal care, and with a normal diet which in the majority of children produces undisturbed and satisfactory development. This unusual response has been explained by assuming the existence of a special predisposition of tissues and organs to normal stimuli. These predispositions to diseases are referred to as diatheses. The predisposition can remain unrecognized for a long time. Only when the appropriate stimulus attacks the child the special hitherto latent predisposition becomes manifest.

Depending on the organ system (skin, mucous membranes, lymph vessels, nervous system) which reacts in a typical manner to normal outside stimuli with pathological symptoms, exudative, lymphatic or neuropathic diatheses with their specific manifestations have been described in these patients. The sharp differentiation of the individual diatheses has, however, not been found feasible. In many infants the diathesis manifests itself simultaneously or successively in several organ systems. The concept of diathesis in its original form as a manner of reaction of a certain organ system has therefore been abandoned at present. It has been replaced by the description of a number of pathological reactions, the pathogenesis of which is only little known. In more recent textbooks of pediatrics the syndromes are described in different chapters usually in connection with the diseases of one organ or organ system (for instance eczema in the chapter on skin diseases). It remains to be seen whether it is correct to so to speak take up again the concept of diathesis, as described by *Czerny, Pfaundler* and particularly by French authors as arthritism. Following the current tendencies, we shall discuss here several frequently occurring pathological syndromes which can be influenced by nutrition and which originate in the different organ systems on the basis of a particular composition or a particular predisposition of the tissues.

The pediatrician will encounter such pathological reactions particularly on those organ systems which are easily accessible to external stimuli. The presence of an abnormal irritability can therefore be frequently recognized on skin, mucous membranes, lymphnodes and the nervous system.

IDIOSYNCRATIC AND ALLERGIC REACTIONS

(*Eczema. .Food Idiosyncrasy*)

Eczema during infancy is today described as manifestation of an allergic condition (allergic diathesis, epidermal-allergic predisposition). The "harmful agent" is a substance which is tolerated by the majority of infants without causing pathological skin reactions and which in only few infants leads to pathological symptoms. The portal of entry or the area of reaction of this substance which is at times known, but mostly of unknown origin, is most frequently the skin, but at times also the mucous membranes of the air passages or of the intestines. The substances which produce the pathological reaction can be exogenous substances which act directly or by contact or occasionally also through the gastro-intestinal tract. Frequently they appear to be bacteria or bacterial products which are primarily present on the skin or which settle rather accidentally on the skin as a secondary infection. The sensitivity for this substance (and this is probably most frequently the case during infancy) can lead to pathological reactions in the predisposed individual, through repeated contact with the same substance by way of sensitization. The pathological process is primarily a serious inflammation which is frequently maintained, concealed and changed by a secondary infection. In other patients the special constitution consists in an idiosyncrasy against one single well definable substance. This includes those idiosyncrasies of the skin which can already occur during infancy, for instance the idiosyncrasy against primroses, or the abnormal reaction toward heterogenous food substances such as cow's milk idiosyncrasy, egg yolk idiosyncrasy, etc.

In describing the symptoms of abnormal reactions of the skin organ it is necessary to consider separately the pathological manifestations in the different age groups. In the different quarters of the first year of life the symptoms vary with regard to form, location and appearance. Some of the abnormal skin reactions during early infancy are not the result of an allergic reaction. This includes particularly the skin manifestations which can be traced back to a deficiency in certain food substances. The unusual (allergic) reaction of the skin occurs to stimuli which at this age appear in the daily routine and with normal care. This applies particularly to the frequently, mostly symmetrically occurring eczema in the face, the changes on the scalp which accompany the eczema and to the more or less extensive eczema on trunk and extremities. If localized eczema appears on the infant, for instance on one cheek, on the forehead, on the buttocks, etc., it is usually possible to find one certain substance as the cause (eczema venenatum). Thus a localized eczema can develop if the mother kisses her child with lips to which lipstick had been applied. Eczema on the buttocks can be caused by sitting on a rug made of animal fur or on a freshly painted chair.

The elementary manifestation of the eczema is a small blister which initially is filled with a clear fluid. These blisters develop as a result of an intercellular edema in the parenchyma of the epidermis. The blisters are characterized by the invasion of lymphocytes into the blister (spongiosis lymphocytaris). When the blister bursts after a short time the fluid spreads over the adjoining skin and a moist eczema develops. The skin which is covered with this escaped lymph is a good culture medium for the skin bacteria which are always present in abundance or for bacteria which find optimal conditions for their development on the moist and damaged skin. In this manner the infected eczema develops, with the redness of inflammation added to the redness of the eczema. After the spread-out and infected fluid dries up the eczema becomes crusty and impetiginous.

Eczema should, because of the different manifestations and different therapy, be distinguished from dermatosis which in contrast to eczema already appears in the first three months of life. This skin disturbance differs from eczema primarily in its etiology. Dermatosis of the first three months of life disappears after six to 12 weeks and in most patients does not recur at any later time. Only in a few infants whose skin is probably constitutionally predisposed or whose damaged skin is secondarily sensitized the dermatosis can develop into a real eczema which then shows no differences from other eczemas with regard to course and duration. The dry and scaly skin disturbances of the first three months of life have been described by pediatricians as dyskeratosis, psoriasoid and seborrheic dermatitis of the first three months of life (*Moro*). In contrast to eczema, which affects only a small number of infants, dyskeratosis occurs in a more or less severe form in many infants during the first months of life, particularly during the winter season. There is no justification in these cases of psoriasoids to assume the presence of a special constitution or a more or less characteristic hypersensitivity.

The differentiation between dermatosis of the first three months of life and the real eczema of the second and fourth quarters of the first year as well as the neurodermatitis at the end of the first year of life is also necessary for practical reasons, because of the entirely different morphological structure of the three diseases, because of the difference in prognosis and because of the differences in therapy.

The pathological symptoms in the first three months of life are erythema and a more or less severe scaling which follows or accompanies the erythema. The initial manifestation of this erythema is a nodule which does not develop further into a blister. Seborrheic crusts develop on the scalp. Scaly areas which are usually arranged in circular or arcade form and which are more or less reddened, appear in the face while the skin surrounding these areas is of normal tender appearance. For these disturbances which occur in a similar form also on trunk and extremities, the term of psoriasoid has been proposed because of certain similarities to a psoriasis. Because of the severe reddening and scaling of the skin it is more correct to speak of a seborrheic dermatitis.

Secondary infections of these skin diseases of the first three months of life occur hardly at all. Even in the most severe forms of scaling where the epidermis might be completely lost, particularly in the mechanically injured areas (buttocks, skin folds on neck, armpit) we observe only occasionally a more severe wetting and only rarely a secondary infection. In this manner bleeding defects can easily develop in dermatitis on the uppermost skin layers which occur most frequently in form of intertrigo on the buttocks or in the skin folds. In any case, intertrigo resembles seborrheic dermatitis much more than the real eczema. This is also shown by the fact that we frequently find changes in the areas adjoining the intertrigo which resemble seborrheic dermatitis. This includes for instance the erythema gluteale; these are round, lentil-sized, papular eruptions which resemble syphilitic lesions and the erythema vacciniforme which resembles the pustules of vaccination.

The most severe degree of erythema and scaling is known as erythrodermia desquamativa (*Leiner*). On the basis of the time of its appearance, the absence of exudation, the absence of secondary local infections, this skin disturbance is characterized as the most severe form of seborrheic dermatitis. In all severe forms of seborrheic dermatitis there is a danger of septicemia caused by the invasion of pathogenic germs through the rhagades which appear on the affected dry skin. Thus some of the children with erythrodermia desquamativa die of septicemia.

The different forms of skin disturbances which belong here can occur in infants in any nutritional state. Neither bottle fed infants nor breast fed infants are spared. The most extensive processes, however, are observed more frequently in children in a bad nutritional state. In severely dystrophic and in atrophic infants seborrheic dermatitis can occur also after the third month of life. Any improvement of the nutritional state diminishes the intensity of the disturbances.

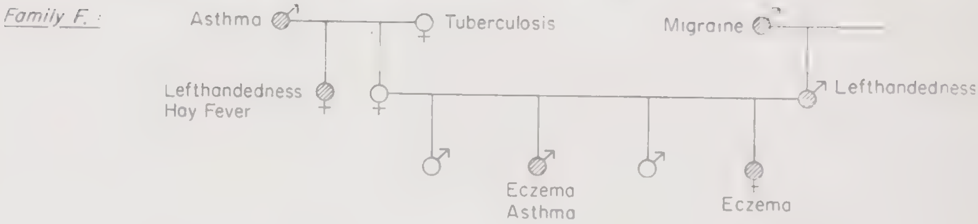
Seborrheic dermatitis as well as other skin disturbances which are caused by primary skin infections do not come under the heading of eczema. This includes impetigo contagiosa which is a primary infection of the skin with staphylococci or streptococci. Skin disturbances which develop as a result of an infection of the skin with diphtheria bacilli and which can closely resemble an infected eczema are of particular practical importance. The greyish-green membrane which forms in those areas where the diphtheria bacillus settles, the tendency to develop deeper ulceration and finally the bacteriological proof of diphtheria bacilli characterize the disease. Staphylomycosis and diphtherial infection can also develop on the basis of eczema.

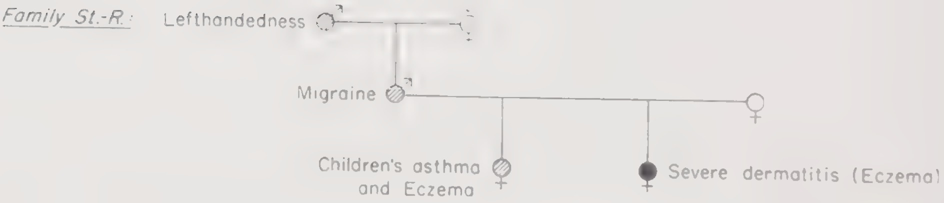
Constitutional eczema appears in almost all cases at a later period of infancy. It is rare during the first three months of life. It appears questionable whether an allergic eczema develops at all before the fourth month of life. Only in a small number of children the scaly erythema, the seborrheic dermatitis, changes gradually in the course of months into a wetting eczema. In another group of infants who also suffered from seborrheic dermatitis during the first months of life eczema occasionally appears as an entirely new disease after an interval during which the skin seemed healthy. However, this eczema does not occur any more frequently in children who had suffered from dermatitis during their earliest infancy than would be expected from experience with a group of infants whose skin had been normal during the first three months of life. And finally there is a group of children who had a clear skin, free of any pathological symptoms, throughout the entire period of early infancy and who develop an eczema only in the second quarter of the first year of life or later.

In the third quarter of the first year of life, at times already earlier, we observe pathological symptoms on the mucosa which in the course of the second or third year of life replace completely the manifestations on the skin. The abnormal disposition of the organism is shown by the tendency to colds and to catarrh of the upper air passages, to chronic bronchitis, frequently of an asthmatic nature, at times also attacks of pseudo-croup and of asthma and recurring attacks of diarrhea. In these children not only the skin but also the mucosa react to the stimulation of normal care with severe exudation. These symptoms of exudation on skin and mucosa induced *Czerny* to apply the well suited term of exudative diathesis for the entity of these pathological symptoms. This constitutes the type of the sensitive child. The simultaneous pathology of exudation on skin and mucosa which frequently occurs at the end of the first year of life does not develop in all children. Some children manifest their predisposition to disease only by changes on the skin while others show the sensitivity of the mucosa with no or very minor skin disturbances. It remains to be proved whether this is only an abnormal predisposition of individual organ systems or whether the stimulus which is necessary for the manifestation of the allergic eczema is accidentally missing, or rather whether, which happens at times, the allergen manifests itself only on one or another of the mucous membranes.

The predisposition to all these diseases is hereditary. By inquiring into the history of the family of the patient it can again and again be confirmed that certain diseases which at first seem to have no connection with the skin disturbance of the infant are found repeatedly in the family of this patient. It appears that the mother and mother's family tend to pass on this predisposition to a larger extent than the paternal family. Among diseases which repeat themselves in the family of the children with allergic manifestations (eczema, asthma, etc.) we observe in siblings and in parents and grandparents or in more distant relatives: asthma, migraine, gout, rheumatic diseases, hay fever, and stones as well as the skin disturbances which recur frequently in the histories.

The following pedigrees illustrate these connections:





Note: Lefthandedness, too, is part of the "arthritism."

Just as the diseases change in the course of generations, the form in which the abnormal tissue quality manifests itself undergoes alterations in the course of the life of an individual. This transformation can be observed already in the short period of infancy. The concept of arthritism of the French school probably describes this fact best.

Eczema develops only as a result of an irritation. In practice, therefore, an attempt must be made to find the cause for this irritation so that it can, if at all possible, be removed from the environment and the care of the infant. A number of children whose facial skin is irritated by an abundant flow of saliva, by vomitus or by constant sucking on the fingers have a tendency to develop eczema which can be placed in the same category with the eczema which is found on the hands of adults and which is caused by the irritation through certain substances; however, this type of "eczema" in infants is actually closer to a dermatitis. An example for the development of eczema due to external irritation is also scabies which in some children produces widespread eczema. But manifestations occur not only through stimuli which meet the body from the outside. It is likely that metabolic products can also act in this manner. It is even possible that products of normal metabolism, too, can cause pathological symptoms in the predisposed child.

Up to now it has been possible to determine the causative stimulus in only a small number of patients with certainty. The recommendation which is customary in the treatment of eczema, not to use water for cleaning face or body, is nothing but the exclusion of an eczematogenous irritation. Many methods which are applied in the treatment of eczema attempt to keep the harmful but not clearly discernible substance or irritation away from the sensitive tissues.

Once an eczema has developed on the skin the skin loses also its resistance against other damages. It becomes a culture medium for bacteria and thus an infected eczema finally develops as a result of the irritation of normal skin bacteria or of secondary bacterial implantation. Infection produces stronger secretion. This transformation of eczema into an infected condition usually begins in the face while eczema on other parts of the body shows much more rarely signs of secondary infection. The infected eczema can turn into a severe, even fatal disease which is accompanied by high temperatures and severe damage to the digestive processes. What is described as death due to constitutional eczema is probably an acute, fatal pyemia or septicemia caused by absorption of large quantities of bacterial toxins from the infected skin. The infection of an eczema develops particularly in the so-called lymphatic children. A greater permeability of the lymph stomata of the skin or a stronger lymph flow in the deeper layers of the skin favors the severe exudation of the eczematous skin. The more abundant the quantities of secreted lymph the more favorable are the conditions for the multiplication of skin bacteria.

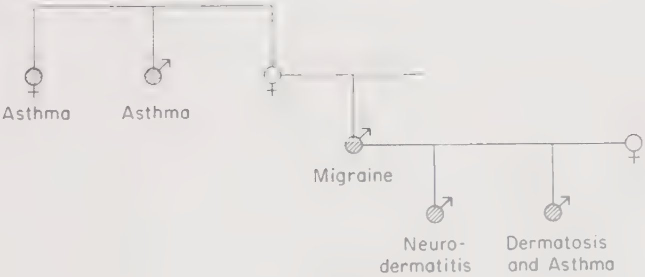
Examination of the metabolism of children with eczema has contributed little to the understanding of the abnormal physical disposition of these children. The nitrogen metabolism proved to be normal. No basic disturbances in the fat metabolism could be observed except for an occasionally occurring poor utilization of the fat. In this respect it appears as yet doubtful whether these changes are not caused by secondary nutritional disturbances. With regard to the carbohydrate metabolism the limit of assimilation for sugar is perhaps reduced so that a glycosuria can develop more easily than in the healthy child. The salt metabolism appears more labile than normally. Chlorine is more easily retained but occasionally also more easily lost than in the normal child. This and the stronger tendency to sodium retention contribute to the fact that the tendency to loose water retention (pre-edema) is particularly great in children with eczema. On the other hand we observed the tendency that even minor disturbances may cause the loss of large amounts of fluids. Children with eczema frequently are hydrolabile. These peculiarities in metabolism, however, offer no explanation for the pathological symptoms of the skin.

It must be assumed that the anatomical basis of eczema is a primary, altered quality of the skin. Constitutional peculiarities of the spongiosa layer of the skin must be made responsible for the development of exudation and of wetting. The appearance of eczema in infants is favored by a lymphagogue, vasomotorically unstable skin. The eczema occurs when an eczematogenous stimulus meets organs which in these children are in a state of "irritable weakness" (*Hufeland*), although it has so far not been possible to characterize more closely this peculiar quality or functional modification of organs and tissues.

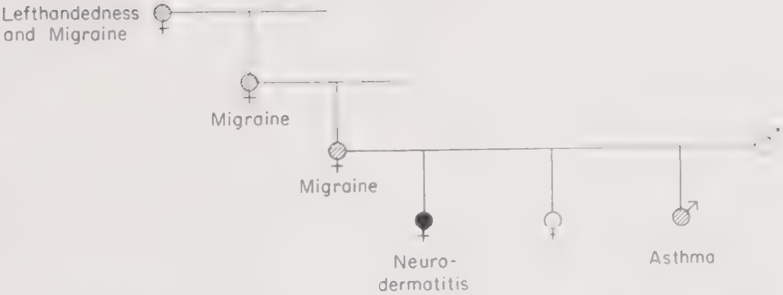
The frequent coincidence with nervous symptoms (itching) also speaks for the presence of a cellular abnormality in the organism since both skin organs and nervous system originate from the ectoderm.

The connections with a special sensitivity of the nervous system are also shown by the presence of other nervous symptoms in the families of these children, as demonstrated by the following pedigrees:

Family P



Family a.B.



In the final analysis, however, it is perhaps not the ectoderm but rather the mesoderm which is the real seat of the disease. The mesoderm participates in the structure of the outer and the inner integument by supplying the basic structure for skin and intestines. This would correspond to the fact that apart from the irritability and weakness of the skin similar changes also develop in mucous membranes and that, as shown by observations regarding the hereditary trends of the symptoms, a large number of anomalies may appear in other organs which are interpreted as "mesenchymal deficiencies." This applies also to the allergic diseases such as asthma, hay fever or disturbances such as migraine and epilepsy.

Many types of eczema occur in the adult, according to the views of dermatologists, as a result of a reaction to a specific irritative stimulus. It is the reaction to an acquired allergy or to a congenital idiosyncrasy toward one or several discernible stimuli. Industrial eczema or eczema caused by primroses are examples for this. Most forms of eczema in adults develop as a result of "reciprocal action of eczematogenous stimuli and predisposition to eczema." In the infant the situation is much more difficult. As opposed to the adult, we can hardly ever discover one single substance which caused the allergic condition. The stimuli which in the child are sufficient to cause eczema are minor and extremely diverse. They are so closely connected with the routines of the customary care of the body or of "normal" nutrition that the child develops an over-sensitivity for daily and frequently unavoidable stimuli. Since in the infant "anything" can produce an eczema it is almost impossible to determine the causative specific stimulus in the individual patient. This explains to a certain extent the difference in theory regarding the pathogenesis of eczema which still exists today at times between dermatologists and pediatricians. In the pathogenesis of eczema in the adult the possibility of definitely determining the eczematogenous stimulus emphasizes the exogenous factor over the importance of the abnormality of the skin in the pathogenetic processes. In the infant, where the eczematogenous stimulus can only rarely be characterized, the predisposition of the skin appears more important. It has already been mentioned that a few forms of eczema in infants originate due to an over-sensitivity for a single stimulus (e.g., chocolate, varnish, lipstick and numerous other substances). In contrast to the adult the results of testing for allergens in infants with eczema are sparse, in fact mostly negative.

The determination of an allergic condition on the basis of some manifestations on skin or mucous membranes does not in any way signify an unalterable unfavorable prognosis. Even with the existence of a predisposition for this disease it happens not infrequently that a child suffers only for a short time of eczema which then disappears and never recurs. It is also by no means to be expected that every patient with eczema will in later life develop a particular susceptibility to asthma or migraine. Only a few children will suffer from these disturbances throughout their later life. The unusual irritability and weakness of the organs and tissues, however, cannot be removed. Despite of this, the prognosis of eczema during infancy may be favorable because most forms of eczema disappear by the end of the first year of life and only a small number of patients will sooner or later suffer from other manifestations of "arthritism."

Treatment of the most frequently occurring skin disturbances of the first year of life will be discussed here only insofar as it can be effected by changes in the diet. Treatment of seborrheic dermatitis is different from that of an allergic constitutional eczema.

The majority of cases of seborrheic dermatitis are today considered as an avitaminosis B, in naturally as well as in artificially fed infants. This applies particularly to those cases which occur in the wet and cold seasons. The factor in the vitamin B complex which causes the dermatitis is the biotin. The proof for these correlations is furnished not only by the curative effect of biotin but also by the fact that breast milk and cow's milk contain less biotin during the winter months than during the summer season. With the discovery of the significance of biotin deficiency as cause for seborrheic dermatitis an etiological therapy became possible which considerably shortens the duration of the disease. While in former years the disease lasted for two to three months and the care of the child was particularly troublesome, the disease now is cured within two to three weeks following the supply of biotin per mouth or in form of intramuscular injections.

In the artificially fed child the biotin content of the diet can be increased by augmenting the protein content of the food or by changing to a less diluted milk mixture, and thus the healing process can be accelerated. In place of half milk the child receives 2/3 milk or whole milk or evaporated milk. Occasionally it is recommended, if more severe diarrhea occurs, to give protein milk for a while. The addition of a calcium-protein preparation (1 to 2% Casec) has a similar effect. Biotin is given to infants in quantities of 5 to 10 mg per mouth or 5 mg intramuscularly per day.

In the breast fed child one to two meals are replaced by cow's milk which is richer in protein and thus in biotin, or here too 1/4 to 1/2 teaspoon of a calcium protein preparation is given with every breast meal, dissolved in a small amount of water or expressed breast milk.

It is necessary that infants during the first months of life who suffer from seborrheic dermatitis show a satisfactory weight gain. Any dystrophy, regardless of whether it occurs in a breast fed child or in a bottle fed child, reduces the resistance of the skin which is already damaged by a vitamin deficiency, against any external irritation. In addition, the diarrhea which is present in many of these patients exerts a constant irritation, particularly on the skin of the buttocks, so that a particularly severe intertrigo develops in these areas. In any case it is wrong to give children suffering from seborrheic dermatitis a milkless diet. Even the omission of a single nutritive substance, for instance fat or protein, is superfluous and even harmful.

Apart from the specific therapy with biotin and the nutritional therapy, harmful stimuli must as far as possible be kept away from the body of the child. Any application of irritating ointments should be avoided. Bathing in water to which bran, talcum or starch have been added, and frequent changing of the diapers is important for these children in order to prevent irritation of the skin and secondary formation of rhagades. Lanolin or a mixture of castor oil, paraffin oil and glycerin in equal parts is effective in the care of the skin. Rubber or plastic pants and rubber sheets should be avoided; loose clothing of the children with as much access of air to the skin as possible promote the healing of seborrheic dermatitis. The facial skin is protected against external injuries by the application of powder or of indifferent ointments such as zinc ointment or zinc oil. It is necessary to remove the severe seborrhea of the head which afflicts many of these children. The scales can be removed by applying a cloth, thickly covered with 2% salicylate ointment, for 12 hours, followed by washing of the head, preferably with green soap, and applying a harmless fat to the scalp (castor oil produces the least irritation). The treatment of the scalp with salicylate vaseline should be repeated only in case of a recurrence of severe scale formation.

Together with an improvement of the skin disturbances the eosinophilia which occurs in many of these young infants, disappears. This eosinophilia which was for a long time considered as a symptom of allergic eczema, is merely the result of a pathological process on the skin, just as eosinophilia can also occur in any other skin disease, for instance in scabies in children.

Seborrheic dermatitis does not always occur during the wet and cold season. In these cases the curative effect of biotin is less pronounced. These forms of seborrheic dermatitis can, however, also be cured by giving a protein rich diet which cures the diarrhea, and by indifferent local applications supported by ultraviolet irradiations.

If a seborrheic dermatitis of the first months of life changes at an earlier or later date into an eczema the treatment of the eczematous dermatitis follows the customary methods of treatment of eczema (see p. 504).

Treatment of the most severe form of seborrheic dermatitis, of erythrodermia desquamativa (*Leiner*) follows the rules established for the treatment of psoriasoid skin eruptions. The symptoms are more severe and more serious. However, the seriousness of the disease seems to have been overestimated in the first descriptions. A child suffering from erythrodermia desquamativa is particularly threatened by the development of septicemia. The skin, which in many places is damaged by rhagades, becomes easily the portal of entry for pathological germs. The youthful age of the patients, the usually reduced nutritional state and the resulting lowering of resistance against all infections favor the implantation and spreading of pathological germs.

Here, too, it is essential for the treatment that the nutritional state of the child be raised. The nutritional therapy for erythrodermia desquamativa is similar to the treatment indicated for seborrheic dermatitis. In these cases, too, it is essential to avoid undernutrition. Even in case of rather severe diarrhea the special nutritional therapy must be given for only a short time. Breast fed children who not infrequently suffer from erythrodermia desquamativa should be weaned only if disturbances of the general condition develop. Diarrhea can be cured by adding suitable feeding mixtures (protein milk, protein calcium preparations, etc.). In general the rules applying to the treatment of diarrhea in breast fed infants should be followed (see p. 136). The replacement of one to three breast meals by artificial therapeutic mixtures might become necessary. In individual cases the physician might be forced to temporarily eliminate the breast feeding altogether. Usually the breast feeding can be taken up again after a short time without danger of a recurrence of diarrhea or of the skin disturbances. It is important that the breasts be pumped in order to maintain the milk secretion. In artificially fed infants the diet is supplemented or replaced by the addition of anti-dyspeptic feeding mixtures. Buttermilk and protein milk have been found suitable for this purpose. In the dietetic treatment of erythrodermia desquamativa an ample vitamin supply has also been recommended. No definite advantage has been observed by the addition of fruit juices.

In treating the skin changes the medications used should be as non-irritating as possible. In order to remove the severe seborrhea of the scalp, which tends to recur, it is, however, necessary to apply once a 1 to 2% salicylate vaseline. The application of a drying powder has been found effective in the treatment of the scaling and inflamed skin. As long as severe diarrhea continues the area of the glutei is protected with zinc paste, zinc oil, cod liver oil ointment or similar ointments. In general the skin can heal best if maceration is avoided as far as possible. This includes the omission of the daily bath. Oil, paraffin oil, and only later bran and talcum baths are used for cleansing the body. As much access of air to the body as possible is desirable. Any tightly fitting clothing should be avoided and the child should be dressed as loosely as possible. The rubber sheet should be removed. In order to expose also the back to the air circulation it is recommended to place the child in a sort of hammock which can be improvised in any children's crib with the help of a sheet and several ribbons or safety pins. In this position, and with the loose clothing of the child, adequate heat supply must be provided during the cooler seasons by hot water bottles or heat lamps. Young, underweight children are best treated without clothing, in an incubator. In order to treat the anemia which occurs in many children suffering from erythrodermia, blood transfusions are necessary. Because of the danger of secondary infection the children should receive penicillin prophylactically, which is given per mouth in children during the first three months of life and later intramuscularly. Other antibiotics (aureomycin, chloromycetin) are also effective. This treatment should be continued until the acute inflammation of the skin and the resulting formation of rhagades have disappeared.

Treatment of allergic or constitutional eczema during infancy is symptomatic since it is possible in only very few patients to discover the causative factor. There are basic differences in the treatment of a non-infected eczema and that of a secondarily infected eczema. This differentiation applies to dietetic therapy as well as to local drug therapy of the eczema. An infected eczema reacts unfavorably to treatment with ointments. Treatment with ointment is indicated in "clean," non-infected or no longer infected eczema. Ointments are a culture medium for the spreading of secondary infections. It is certainly no accident that pathological germs such as diphtheria bacilli and others frequently appear on infected eczema which had been treated with ointment over a long time, so that skin diphtheria or diseases of an impetiginous nature secondarily cause changes and aggravations to the eczema. Nowadays it is particularly advisable to attempt in every case of stubborn infectious eczema clarification of the nature of the secondary infection. Depending on the type of bacteria it is now possible to accelerate the healing of the disease by a specific therapy (antibiotics). If diphtheria bacilli are found, treatment with adequate quantities of penicillin suffices since the bacilli are most likely not virulent. Only in more severe necrotizing processes, which are usually accompanied by fever and disturbances of the general condition, an intramuscular injection of 5000 to 10,000 I.U. of diphtheria antiserum is indicated. The treatment with a suitable antibiotic is recommended in all cases of secondarily infected eczema.

The aim of non-specific local therapy is to change the culture medium in such a manner that the living conditions become unsuitable for the pathological germs which had implanted themselves on the eczema. This can best be accomplished by wet applications with aluminum acetate, 3% boric acid, etc. This brings about an improvement in the acute inflammation, a reduction in the secretion and a drying of the skin. The applications should not be covered with a waterproof cloth. They should be changed very frequently, about every one to two hours. If the wet cloth remains on the body for a longer period of time the warmth under the compresses produces maceration of the skin and through the absorption of bacterial toxins we observe not only an aggravation of the skin disturbances but also high temperatures and severe illness. The innermost layers of the compresses should be replaced at the hourly changes. They bring about a recession of the inflammation and infection already after 24 to 48 hours.

With these methods of treatment (antibiotics and wet applications) it is possible within four to five days to change the infected eczema into a dry eczema. The danger of recurrence remains. Minor irritations suffice to bring about a recurrence of eczema and infection. These relapses are promoted by a severe itching which affects many of these children. Taking care of this itching is therefore part of the treatment of infected eczema. Sedatives (luminal) must be used liberally. Scratching must be prevented by careful handling of the child and by immobilizing the arms.

It is not possible to set down a routine treatment for dry eczema. Even the experienced physician cannot proceed without a certain amount of intuition and, it must be admitted, also some experimenting. This uncertainty is caused by the fact that the skin of every child reacts differently at this stage to any kind of treatment. This does not only apply to the drugs which are added in order to promote epithelialization. It applies also to the basis of ointments. Thus we find some children whose skin will not tolerate mineral fats such as vaseline, while fats like lanolin do not cause renewed irritations. It is therefore advisable to test any new ointment and any new medication at first only on a small skin area before the entire affected space is treated. Tar ointments have been found most suitable at the stage of a non-infected, perhaps still only slightly wetting eczema. A 3% Tumenol zinc ointment or a 2% Tumenol shake lotion is best used in places such as the face where the application of a bandage creates difficulties in infants. For epithelialization and to stop the itching painting with Pix lithanthracis is most effective. The tar is applied once in a thin layer and then covered amply with talcum powder. This produces a covering layer which is not renewed or removed until it peals off after a few days. If there are still any new reddened and slightly wetting places the painting is repeated. If remnants of the infection still exist mild mercury ointment, bismuth-zinc paste, Ichthyol-zinc paste or Ichthyol-shake lotion can be tried. In case of more severe scale formation 5 to 10% sulphur ointment can be used. Mercury should always be used with special caution in case of larger eczematous areas since the absorption of mercury leads in some children to symptoms of poisoning or to allergic symptoms (fever, measle-like exanthema). At this stage of the eczema, when the skin is still slightly reddened and infected, the patience of parents and physician is sorely tried because of the tendency to renewed secondary infections and to recurrences of the eczema. Once the eczema has reached the dry stage it is more likely that it can be controlled by careful handling, by keeping external irritation away and by applying indifferent ointments (lanolin) and pastes to which a tar preparation (Tumenol, Lenigallol, Naphtalan etc.) has been added.

The drying up of the infected skin is supported by measures which have an "internal" drying effect. Two methods can be applied for this purpose: first by changing to a dry diet whereby the child receives only up to 75cc of water per kilogram of body weight with his diet. By feeding the child with thick cereals, by replacing the previously given milk mixtures with correspondingly smaller quantities of whole milk with 15% sugar and by adding thickened vegetable and fruit purées, it is possible to effect a drying up without reducing the caloric intake. This procedure can be applied only in older infants and under climatic conditions which do not require a high fluid intake. During the hot season, in children with fever and in children during the first months of life there is the danger that this procedure might result in damages due to dehydration. Water is withdrawn from the tissues also by a reduction in salt and carbohydrate contents in the diet. By avoiding these water retaining substances dehydration of the tissues develops. This is the basis for the eczema soup (*Finkelstein*), a salt and sugar deficient food which occasionally has been used successfully in the treatment of infected eczema.

Eczema soup consists of the milk curds obtained by precipitation with lab ferment and rediluted with water and small amounts of whey, and sweetened with sugar.

The water losses which take place in this manner manifest themselves in form of the considerable weight losses which occur after changing the child's diet to eczema soup. The severity and speed with which this dehydration takes place is not without danger in younger and in hydrolabile children. If no salts at all are given a dehydration can develop which exceeds the desired degree. The special eczema diet should be applied only within the frame of hospital supervision. A dry diet and eczema soup are incomplete foodstuffs and may be given for only a short period of time. They are occasionally effective in the first phase of a severely infected eczema.

In older eczematous children those substances should be omitted from the diet which are known to produce frequently eczema in infants. This includes the protein of the milk, the milk fat and eggs, particularly egg white. Therefore sweetened almond milk, which however has a biologically very incomplete protein, or soybean milk is recommended as basic food. Older infants at the end of the first year of life, who are good eaters, can also be fed at the beginning of the dietetic treatment for one to two days with only fruit juices, puréed fruit (bananas, apples) and puréed vegetables. After some days this milk free diet is supplemented, depending on the age of the infant, with boiled sweetened rice, farina, puréed lean meat, liver, mashed potatoes (prepared with water) or zwieback (soaked in almond or soybean milk). After some time an attempt can be made to begin the replacement of a few meals with half milk. Commercially prepared types of milk which contain little salt have also been recommended. The reaction of the skin must always be carefully observed. If the first meals are well tolerated the milk can be given more frequently and in stronger concentrations. Since the sensitivity of the skin toward external and internal stimuli diminishes after the first year of life, many children can during the second year of life be placed on an approximately normal diet which also contains whole milk, egg, etc. Caution is indicated with regard to the intake of animal fats, particularly in those patients where the eczema itches considerably or who develop a tendency to lichenification and those who at the end of infancy show a transition to neurodermatitis.* For these patients a fatless diet (skimmed milk, no egg yolk, butter, cocoa, cod liver oil, etc.) is indicated. The lacking fat is replaced by an increase in the quantities of carbohydrate.

This dietetic treatment has been found effective particularly in fat, pale children suffering from eczema. The incidence of eczema in skinny children is lower than in obese children. In these poorly nourished, underweight children we observe more frequently changes in the skin which resemble the scaly dermatitis which only occasionally becomes secondarily eczematous. In the skinny or emaciated patients it is necessary to bring about a gain in weight. The primary and secondary skin changes disappear only with an improvement in the nutritional state. With a reduction in animal fats an abundant supply of protein (skimmed milk, white cheese, meat) is necessary. Vegetable fats (margarine) in moderate quantities are well tolerated by these children.

An attempt should always be made to discover the source of irritation which caused the eczema. This search, however, frequently remains unsuccessful in infants. But even in the infant we find at times a pronounced allergic reaction of the skin caused by a certain allergen. However, definite allergic reactions are usually discovered only in older infants and young children. The value of testing for allergens is therefore very low during the first nine months of life.

Allergic and idiosyncratic reactions in the infant do not appear only on the skin. Occasionally the over-sensitivity manifests itself in form of pathological reactions in the gastro-intestinal tract. The interpretation of these symptoms, which at times are very severe, is frequently difficult in the beginning. The issue is clarified by the anamnesis or by the onset of reactions with every recurrence of a certain irritative stimulus. The so-called cow's milk idiosyncrasy (weaning dyspepsia) and an idiosyncratic reaction to certain food substances, e.g., egg yolk or bananas, are among the reactions of over-sensitivity in the sphere of nutritional processes. These allergic and idiosyncratic reactions can at times take a rather stormy course.

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* Dermatologists tend to differentiate between eczema and neurodermatitis. In contrast to the eczema, whereby the epidermis is the primary area of reaction, the neurodermatitis is claimed to be the result of a "cuto-vascular predisposition." The tendency of neurodermatitis to develop into eczema is admitted. However, the histories of the disease and the families of infants suffering from eczema or neurodermatitis, respectively, show that there must be a correlation between the two diseases which can perhaps be explained with the development of the skin organ.

Allergic conditions against cow's milk occur in infants who for some reason received cow's milk mixtures as newborns for only a short time or even for just one or two meals, who were then fed on the breast for a longer period of time and who at the time of weaning develop acute diarrhea with considerable weight losses. It is probable that the newborn whose intestines are still passable for heterogenous protein, develops an allergy against cow's milk. If after several months cow's milk is again given for the first time the child shows an allergic reaction which occurs in form of an acute diarrheal disturbance. These conditions have been described as weaning dyspepsia.

Aside from these there are a number of children with an apparently primary idiosyncrasy against cow's milk. These infants react violently to the first cow's milk feeding with allergic shock, diarrhea, vomiting and weight losses. The efficacy of antibiotics against these forms of diarrhea is uncertain since they can cure only a secondary invasion of the irritated intestines. If the weight losses are great infusions of salt or glucose solutions or plasma are indicated. If the condition becomes dangerous the infusion of these solutions with the addition of hyaluronidase, which is technically simple and can be carried out rapidly, is recommended. The sensitivity against cow's milk disappears in the course of the second year of life. It is possible to gradually overcome this state of over-sensitivity by adding initially a few drops of cow's milk and then gradually increasing these quantities. During the period of transition following weaning from the breast it is recommended to give a milkless diet, with the protein supplied, depending on the age of the child, in form of amino-acid hydrolysate, meat or liver and with the addition of vegetables, fruit, rice, lentils, sugar, white bread, etc.

The idiosyncrasy against egg yolk can perhaps best be illustrated by the following case history:

Child—A.M., seven months old, is admitted in a severely ill condition, grey and cyanotic, with high temperatures, diarrhea and vomiting. Following an infusion and careful feeding with protein milk in increasing quantities, a strikingly rapid recovery and weight gain are observed. During the third week of hospitalization suddenly the same dangerous condition occurs from which the child recovers again just as rapidly. Only after a third recurrence it is discovered that the severe reactions occur every time egg yolk is re-introduced in the diet. Questioning also revealed that the first attack took place when the child received egg yolk for the first time in his life. After excluding egg yolk from the diet for nine months it was given again in minute quantities which were gradually increased. At the age of about three years egg yolk was tolerated without reaction. However, an aversion against eggs persisted in the child.

LYMPHATISM

Although *Czerny* believed that the lymphatic constitution, which was first described by *Virchow*, could be included in the exudative diathesis or in the allergic diathesis, it seems more practical to regard lymphatism as a separate physical condition. This point of view seems justified because children have been observed with the typical symptoms of lymphatism who do not show any signs of an allergic constitution (allergic or exudative diathesis) either in their histories or in their further development. On the other hand the two constitutional anomalies frequently occur side by side. There is also a close connection between lymphatism and neuropathy so that the term neurolymphatism has been evolved which in many points resembles the neuroarthritism described by French authors.

The lymphatic diathesis manifests itself during infancy only after the sixth month of life. The nature of the diathesis, according to the original concept of *Virchow*, consists of a hereditary tendency of the lymphatic tissue to react to relatively minor irritations in a "pathologically aggravated manner." Damages to the corresponding lymph region are answered with severe and prolonged swellings of the lymphatic tissues. But just as the nature of exudative diathesis or of the allergic condition is not covered by a description of eczema, the lymphatism is not only an abnormal tendency to pathological reactions of the lymphatic tissues. Here, too, we find deviations from the normal in all cells and tissues of the body.

Children suffering from lymphatism lack the good turgor of the eutrophic individual. The tonus and the tension of the musculature are absent. The usually abundantly developed adipose tissue is "different." The fat is soft and spongy and pasty. The face and the backs of hands and feet seem swollen and edematous, although these skin changes are not the result of an abnormal water accumulation. Skin and mucosa frequently appear pale although there is no anemia. There is a pseudo-anemia caused by a narrowness or narrowing of the capillary system. Even during infancy some of these children are phlegmatic and particularly quiet. Their psychological development can be delayed. The children never belong to the typus cerebralis, more frequently to the typhus digestivus. The body structure is compact, the neck is short, the abdomen large and protruding, adipose tissue is abundantly developed on the large, frequently hanging cheeks as well as on the lower part of the body, the buttocks and the upper thighs.

The unusual quality of the tissues manifests itself in the tendency to abnormal glandular swellings even from very minor stimuli. We find swelling of the tongue follicles, enlargement of the spleen and perhaps also of the thymus. The lymphatic ring of the pharynx is also stronger developed at the end of the first year of life than would correspond to the child's age. The posterior pharyngeal wall is strongly granulated and adenoids cause difficulties in breathing through the nose even during infancy. Among these pathological symptoms particularly the tumor of the spleen which can be found by palpation, can cause diagnostic difficulties in pale children. Anemia and other diseases of the blood, tuberculosis and syphilis must be excluded in every case.

Lymphatic children show a predisposition to a number of diseases. Rickets and tetany occur more frequently in lymphatic than in normal infants. In such cases rickets develops particularly in the long bones. Severe enlargements of the epiphyses and bowing of the bones are not infrequent. The tetany of lymphatic children is characterized by severe laryngospasms. The tissue of the lymphatic individual proves very unstable toward any kind of disease. Any nutritional disturbance and any febrile disease is accompanied by great weight losses. Any infectious disease is characterized by its severity. In all kinds of infections these children develop particularly high temperatures.

An unusually large thymus gland should also be included in the picture of lymphatism. Its hypersecretion was regarded as cause for sudden death. This theory can no longer be maintained today after it has been found that enlarged thymus and enlarged lymph nodes occur regularly in infants who suddenly die without having shown any previous signs of disease. In any case, the sudden death of an infant cannot be explained with the finding of an enlarged thymus gland or with a hyperthymization of the organism. In the final analysis the cause of sudden death of apparently healthy infants who are usually beyond four months of age, has not as yet been clarified. Interstitial pneumonia, asphyxia, aspiration of vomitus etc. are not sufficient in every case to explain an unexpected death in children of seemingly good health. We have repeatedly observed histological changes of the adrenals in these children. However, sudden death occurs more frequently in fat, pasty looking children than in normal infants. Excitement and fear seem to promote the occurrence of sudden cardiac arrest. Superfluous diagnostic or therapeutic measures should as far as possible be avoided in pasty looking lymphatic children.

In predisposed infants lymphatism is promoted by a wrong diet. Overfeeding, one-sided nutrition particularly with milk, flour and sugar, further the development of the lymphatic pasty habitus. With the same incorrect diet the constitutionally normal child remains sturdy and solid in his structure. The lymphatic child becomes spongy, pale and pasty. The swelling of the spleen is also in part a result of overfeeding in these obese children. If common infections attack such a predisposed child the swelling of the spleen can develop also in diseases where this symptom is usually absent in the majority of children.

Even without excessive overfeeding or one-sided incorrect nutrition a predisposed child develops the appearance of a lymphatic individual, that is, the soft spongy fat and the pallor. With a suitable diet it is possible to suppress part of the symptoms in the lymphatic child. A diet barely sufficient in calories is indicated, with no weight gains for a number of weeks. This reduction can best be accomplished by decreasing the number of meals. Four meals during the second quarter of the first year, three meals beyond the sixth month of life are adequate. The customary milk and sugar diet should be replaced at an early date by a mixed diet. The daily milk intake should not exceed 200–300g. Beginning already with the second or third month, the children receive a diet which is rich in vegetables, fruit and egg yolk. Any unnecessary fluid and salt intake should be avoided. For children with a particularly pronounced tendency for the development of lymphatism milk is at times completely eliminated from the diet. The children are then fed without milk, with puréed meat, liver and amino-acid preparations which supply the necessary protein quantities.

HYDROLABILITY

Hydrolability is the quality of the organism to react to minor damages with unusually large weight losses. This abnormal reaction is more often a congenital, more rarely an acquired characteristic of the infantile organism. The congenital tendency to abnormal water losses is usually connected with the ability to rapidly regain those weight losses. This up and down of the weight curve supports the theory that the water which is so easily lost and just as rapidly retained originates essentially from the intercellular fluid depots. There are no losses of intracellular fluid which are accompanied by damages to the cells. The weight curve of these infants shows a lack of consistency.

The acquired hydrolability develops in every dystrophic and atrophic infant. The deficiency in the ability to normally retain and bind water makes the curing of any disease more difficult in those children who are damaged in their nutritional state.

Congenital hydrolability usually manifests itself only beyond the third month of life. One single instance of vomiting, one day with fever, a few somewhat loose stools suffice to cause in the apparently eutrophic infant weight losses of 200 to 500g within 24 hours. The children do not look well, they appear pale in the

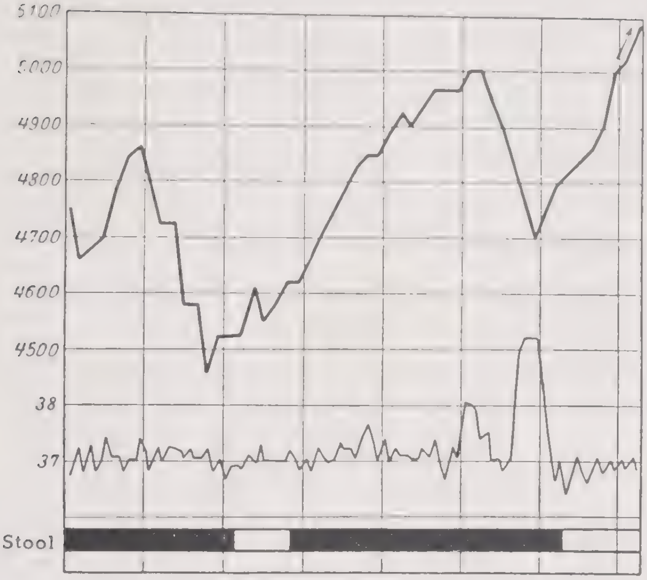

Fig. 61. Marked fluctuations in weight of the hydrolabile child. Slight diarrhea or mild infections lead to marked drops in body weight which improve spontaneously.

face, rings appear around the eyes and the mucous membranes of the lips become strikingly red. Other symptoms of dehydration are absent or are only vaguely indicated. If the damage which caused the water losses disappears rapidly the child will within another 24 hours already regain a considerable part of the lost weight, even without changes in the diet. The appearance of the children improves. After a few days the children seem healthy again until after some time another minor disturbance causes a similar break in the weight curve. These hydrolabile infants frequently are of the typus cerebralis.

Hardly anything is known regarding the cause for the loose water retention in the tissues. Variations in the fluid content of the blood have been reported. It is interesting that the daily physiological variations in the weight curve are particularly great in these children. The daily weight gain of the infant is the result of an increase during the hours between morning and evening (100—200—300g) and an almost similar decrease in weight during the night. The daily weight gain of about 25 to 30g is the result of a somewhat larger increase in the course of the day as against the rhythmically following weight decrease during the night. The daily weight increase in the hydrolabile child is the same as in the hydrostabile child, as long as his abnormal condition does not manifest itself. This resulting increase, however, is reached with considerable daily up and down variations in the weight curve (300 to 600g). This applies also to older children and adults who always look "badly" in the morning and begin to look thriving only in the afternoon and evening (evening beauties).

Prophylactically a diet has been recommended for these children which is particularly rich in lipoids (e.g., egg yolk). These infants frequently confront the physician with difficult decisions. The situation is particularly difficult when the abnormal weight loss occurs for the first time. Once an infant is known to be "hydrolabile" it will become easier to forego any basic dietetic measures, mainly the starvation periods and reductions in food intake. If the weight losses are accompanied by diarrhea and disturbances in the general condition a special therapeutic diet and a gradual resumption of nutrition is unavoidable. In case of severe weight losses and signs of dehydration infusions might be indicated. Care should be taken, however, that only those fluids are used for infusion which do not in themselves promote a loose water absorption in the tissues. Infusions of plasma and amino-acid hydrolysates are more appropriate than salt and glucose solutions.

THE NERVOUS CHILD (NEUROPATHY)

Already during infancy reactions of the nervous functions exert an influence upon the nutritional results. Influences of this nature have already been mentioned at several places in this book. Influences on the part of the nervous system cause difficulties in nursing. This also applies to difficulties which are encountered in some children with any attempt to introduce a new food of a different consistency or taste. It further applies to the tendency in some children to a persistently increased peristalsis, to regurgitation and to vomiting. The lability of the vegetative centers also plays a role in the development of toxic gastro-enteritis. On the other hand pathological symptoms in the nutritional process are frequently considered as "nervous" because no better explanation can at first be found. If for instance a child suddenly loses his appetite without any apparent reason and refuses to eat at all or to eat any solid food this child will be stigmatized as "nervous" by relatives as well as by the physician. Whether this explanation applies in every case must seem rather doubtful in view of the lack of almost any knowledge regarding the psychological processes in the infant. In these cases we apply the term nervous without knowing anything about the deeper processes.

If we observe in an infant an increased excitability, an increased exhaustibility and a reduced ability to return to a state of balance, the functioning of the nervous system can be regarded as an expression of a nervous constitution. The increased excitability affects the entire motor-sensory and vegetative nervous apparatus. Minor irritations affecting the sensory organs, particularly eyes and ears, bring about violent reactions. But even the functions of the organs supplied by the vegetative nervous system appear to be more noticeable in these children in form of discomfort or pains than in the majority of "normal" children. The over-excitability of the vegetative apparatus leads to pathologically increased activity of those organs which are supplied by it. Restlessness, crying, increase in peristalsis are in this sense manifestations of neuropathy. Prolonged restlessness and disturbances of the well being which occur as a result of minor optical and acoustic stimuli and which in the normal child produce only a very temporary minor reaction, are part of these manifestations. The nervous child lacks the instinctively controlled correct and appropriate evaluation af a stimulus or, as *Heubner* once described it, all the stimuli acting upon a nervous child appear increased and distorted as through a magnifying glass. In this manner the abnormal, exaggerated response to a stimulus can be explained. The "nervous" child lacks the "instinctive self-control" of his nervous activities. Fearfulness and superficial sleep are the earliest signs of neuropathy in the infant, as was shown already many years ago by *Oppenheim.*

Appetite and the reaction of the gastro-intestinal tract in the nervous child are of special significance for questions of nutrition. In these cases even the physiological stimulus of food causes exaggerated and inappropriate reactions. All nerve controlled physiological processes are pathologically increased. The manifestations of neuropathy appear most clearly in the digestive processes of the breast fed infant. In the artifically fed child it is not always simple to exclude damages of another nature which were caused by the artificial food itself, as being the cause of the pathological symptoms. But the condition of the nervous system of the child also plays a considerable role in the development and course of nutritional disturbances appearing in artificially fed children.

Is it possible to recognize the nervous child from the form of his body—even at a time when manifestations of the nervous constitution have not or not yet appeared?—A large number of neuropathic children belong in the category of the typus cerebralis (according to *Sigaud's* classification). They are usually children with long and thin limbs, with a long, narrow face, large skull and broad, frequently somewhat protruding forehead. In many cases signs of the asthenic habitus can be discovered so that the appearance of the typus cerebralis is more or less intermingled with features of the typus respiratorius. The hair growth of the children is often abundant from the time of birth. The hair on the head is usually thin and wavy. The so-called *Freund's* crest of hair, the wave resembling the comb of a cock in the center of the skull together with less hairy occiput and temples are observed in this type of children. A severe motoric restlessness characterizes the children. The face is in constant motion. The forehead is wrinkled, the naso-labial folds are taut, the eyes are wide open. But the musculature of the remainder of the body also is in constant motion. Beginning with the first days of life these children frequently assume characteristic forced poses which persist for a long time. Thus some children sleep on their side, with the head and back bent sharply backward. These forced positions frequently remind the observer of the reflex positions which could be produced arbitrarily by *Magnus* and *Sherrington* in decerebrated animals.

The first manifestations of nervousness appear already in the newborn. A number of difficulties develop in the nursing process. The sucking on the breast and the regular development of the sucking reflex do not take place with the same degree of regularity as in the normal child. These conditions have been discussed in the chapter on breast feeding. The difficulties connected with the introduction of the nursing procedure are increased by the fact that the neuropathic child is always born in a nervous environment. Once these earliest obstacles in feeding are overcome with more or less difficulties the development is disturbed by regurgitation and vomiting and by an increased peristalsis which causes frequent eliminations of small quantities of stool; these symptoms increase the nervousness and worry of the child's family. By appropriate measures and with patience it is possible to bring these nervous pathological manifestations to a stop. However, the carrying out of these measures which in themselves are quite simple, offers difficulties to the nervous environment of the child. Some of the orders are carried out only in part. Suggestions on the part of outsiders in matters of care and nutrition of the child are listened to and applied with pleasure. Thus it happens that minor deviations from the normal feeding process which in the healthy child are straightened out easily, develop in the nervous child into serious dystrophic conditions, even in breast fed infants. It would, however, be wrong to wean a child from the breast in such a case.

New difficulties present themselves when weaning of the child is attempted. Getting accustomed to the bottle can become quite troublesome. Cunning and skill on the part of physician and nurse are necessary in order to overcome these difficulties. If the consistency of the food is changed, for instance if cereals are introduced, or if the child is confronted with new food substances and new tastes, for instance fruit or vegetables, new difficulties develop in the nervous child which can be overcome only with patience, perseverance and educational talent on the part of the nursing personnel. It seems, however, not justified to classify all these children as constitutionally nervous. In many cases they are temporary symptoms which are rapidly overcome and which occur in children who neither before nor afterwards show any other signs of neuropathy. However, in a small number of infants it is not possible, despite the skill of the nurse, to overcome the dislike for a new type of food for a long period of time. Those are the children where every meal turns into a struggle and a test of strength between mother or nurse and the child. Another time the resistance of the child might be induced by an attempt to offer a vegetable meal. Frequently this dislike does not remain limited to this one dish but extends to any type of food which is offered the child. If such children are observed during mealtime, we can differentiate between two types. Both of them show a dislike. Both types have a negativistic attitude toward food. One group refuses the food actively, that is, the child struggles, cries, spits and vomits upon every attempt to place some food into the mouth or to swallow it. The other group shows more passive resistance. The children lie still, they also allow the food to be placed in the mouth without resisting, but they do not swallow the food. When the mouth is full they let the liquid and semi-liquid food flow out of the mouth without even moving the tongue. All these children create the impression that the natural pleasure which is caused in the normal child by the intake of food is lacking and that in place of this pleasurable feeling we observe displeasure. Here, too, an external stimulus seems necessary in many cases apart from the endogenous predisposition, in order to bring about these manifestations of neuropathy. Minor disturbances can spoil the appetite in these children. An uncomfortable position of the child, excessively hot food, an unpleasant taste, and other incidences which the healthy child hardly notices or forgets after a short time, remain with the neuropathic child, appear to him enlarged and bring about more serious consequences. A pain upon the introduction of the spoon into the mouth, the touching of a sensitive tooth which is just breaking through, can deprive the child of his appetite for weeks and months. Frequently it is not possible to discover the original cause. Background and care of the child are of little significance. Similar difficulties appear in infants receiving good care in a private home, in infants raised in a poor family or in a child growing up in the collective care of an infant home. All these feeding difficulties disappear some day. It would perhaps be most convenient to ignore them. However, the regurgitation, vomiting and refusal to swallow leads in these children to a deterioration of the nutritional state or to nutritional disturbances. Children who do not want to eat and to swallow soon develop quantitative and qualitative hunger. In addition, the particularly pronounced restlessness and the crying of these nervous children suffice to raise the nutritional requirements of the child by 100% and more during the time of restlessness.

The mechanism which leads to this peculiar behaviour of the children can perhaps be explained by the development of the so-called conditioned reflexes as described by *Pawlow*. Apart from reflexes occurring on the spinal reflex arch other reflexes can easily develop in human beings and in animals which do not take their course over the deeper sections of the central nervous system but which show cortical reflex arches, usually over the optic or acoustic centers. Such a reflex is observed for instance in the following experiment: a dog fed for some time only upon the appearance of a certain color or a certain sound will after a while begin to secrete gastric juices, even without food intake, merely upon the appearance of the color or the sound. The child, too. develops such reflexes at an early age. Thus the hungry and crying infant who is used to having his diaper changed before being placed on the breast stops the crying already when he is changed and freshly dressed. The development of such conditioned reflexes occurs particularly easily in the nervous child. One single painful experience, for instance, is remembered by the nervous child and very soon one phase of the unpleasant experience suffices to bring about the entire reaction.

Heredity and environment appear to contribute in equal measure to the manifestation of irritability in the nervous system. The environment, the nervous and worried parents and other relatives all contribute to the appearance of neuropathic symptoms. The possibilities of errors in treatment, care and education of the nervous child are numerous in such an environment. Any single error suffices to produce pathological symptoms. There is hardly a mistake which is not made by the people attending to a nervous child. However, the recognition that a nervous child is always surrounded by a nervous environment is not only of etiological interest but therapeutically also of practical significance. The first minor manifestations suffice to alarm the nervous environment of the child and to increase the tension. The great amount of activity and polypragmasy, the change in nurse and physician which then take place, again reflect on the child. The symptoms become aggravated and give rise to more errors and irregularities in education and nutrition. It thus happens that finally disorder prevails instead of order and thus disease and sorrow enter the children's room, with nervous children attended to by nervous parents. In this manner severe nutritional disturbances and severe diseases develop already due to misinterpretation of physiological processes of nutrition and due to harmless nutritional disturbances.

The environment is also of importance for the prognosis of pathological symptoms which belong in this category. With reasonable guidance and education a large part of nervous pathological manifestations can probably be avoided. In any case it is not necessary that every child undergoes the long succession of these pathological symptoms customarily seen in these children and so harmful to them. This applies primarily to the processes of nutrition. If the nature of these disturbances is correctly evaluated they should not in any way affect the development of the nervous child. An attempt must be made in children who are known to be neuropathic to correctly evaluate the pathological symptoms which influence the development and to avoid their manifestation by excluding all external damaging factors.

Neuropathy also influences the prognosis of any infectious disease and of any nutritional disturbance. In case of disease a nervous child is always at a disadvantage compared to a child with a well balanced nervous system. The pronounced restlessness of the child, the tendency to the development of diarrhea and vomiting disturb and delay recovery. The difficulties in carrying out the physician's orders which are already encountered in the older infant, the excitement caused by the appearance of the physician, the vomiting which accompanies any throat examination and the prolonged crying which occurs with any kind of examination considerably aggravate the condition of the nervous child as compared to the healthy infant who shows calmness and discipline. The neuropathic predisposition causes an aggravation in the prognosis not only in the acute stage of a disease where such children suffer more and their care is made more difficult. Numerous complications such as convulsions, circulatory failure etc. endanger these children more than the "normal" children. The less favorable prognosis also continues far into the period of convalescence which lasts longer in the nervous child and which is not infrequently interrupted by unpleasant relapses.

Treatment of manifestations of neuropathy which appear in form of localized diseases or pathological symptoms have already been discussed in some of the earlier chapters. An adequate, reasonable and calm attitude can best asure success in treatment. Any sign of indecision and nervousness on the part of the physician, continuous changes in the orders and similar upsets create further severe damage to patients as well as to relatives.

The surprising results which can at times be obtained by a change in surroundings of the neuropathic child show the significance of the environment in the treatment of pathological symptoms caused by nervous disturbances. In the patient's home the requirements are frequently lacking which are necessary for suppressing or curing the nervous symptoms. For the person charged with the bringing up of neuropathic children must possess certain qualities which are not present in everybody who feels called for this task. Equanimity, calmness and composure must form the basis of education already in earliest infancy. Too much knowledge and at times even a high degree of intelligence spoil in parents and nurses the stability in all measures of body care, nutrition and psychological influence. Those mothers and nurses who understand to bring comfort and calmness to their environment are best suited for the care of such an infant. For even the young child already possesses a surprisingly sensitive feeling for the personality and the nature of the person who attends to him and feeds him. Creating an atmosphere of calmness and comfort, however, does not mean spoiling the child, just as regularity in the care of the child must not deteriorate into exaggerated anxiety which burdens the mother with unnecessary work and worry and disturbs the calmness and sleep of the child. Restlessness on the part of the attending person, however, does not only cause discomfort in the child. Faults in the feeding technique, unnecessary changes and irregularities in the choice and composition of the feeding mixtures occur very frequently in homes where we observe restlessness instead of calmness and anxiety and exaggerated worry over the child instead of comfort and composure. Unfortunately the negative qualities which are unsuitable for the care of nervous children are found particularly in parents of neuropathic children. Unsteady, hasty and worried mothers frequently confuse essential and unessential factors in the nutrition of the child. This also creates a difficult situation for the physician. His recommendations are only partly listened to and only partly carried out. If his orders fail to show an immediate success different methods of nutrition are tried. The result is a frequent change of physicians until the child is finally one day taken away from his harmful environment and placed under the care of an institution or a nurse who can without undue difficulties guide the development of the child.

With regard to treatment we must remember that in cases of neuropathy therapy must be instituted not only for the patient but also for his environment. Frequently an improvement can be effected only when the physician who is consulted for the treatment of the child manages to influence the neuropathy of the parents, primarily of the mother. Anxiety neuroses over the growth and development of the child which occur in young mothers, even in those who are otherwise not nervous, are particularly frequent and pronounced with the first child. Worries as to whether the child has enough to eat, whether he gains sufficiently in weight, whether the milk is good, whether the eliminations are too frequent or too rare, and many other problems torment and upset the young mother. Frequently the physician requires all his energy in order to help in such cases. It is above all important that the mother gains confidence in the therapy of the physician. This is possible only when the mother can be convinced of the results of the treatment by a rapid success. The first real success is the weight gain of the child or the cessation of regurgitation and vomiting or the termination of a diarrhea. With the disappearance of the troublesome symptoms in the child the neurosis of the mother can also be brought to an end. This eases the situation considerably. There are sufficient means and ways which make possible an improvement or a cure of these manifestations of neuropathy. Reference might be made here to the chapters concerning the treatment of nervous disturbances in breast fed children, concerning the treatment of regurgitation and vomiting, the treatment of diarrheas in breast fed infants and of diarrheas without disturbances of the general condition. It only remains here to discuss the therapy of anorexia and the treatment of the feeding difficulties which are encountered in infectious diseases.

Everything and anything has been recommended for the treatment of anorexia which usually develops in the second half year of life following the introduction of new types of food or of new tastes. Forced feeding is never to be recommended. At times already a hunger period of 12 to 24 hours and more suffices to overcome the child's resistance. However, these methods are only rarely successful.

Following the period of hunger small quantities (about 100g) of more strongly diluted food should be given. Feeding mixtures which are rich in fats should be particularly avoided. At times it becomes important to institute changes in the manner in which the food is given. For instance, instead of the bottle the child receives the food with a spoon, or instead of the spoon he takes it directly from the cup.

Forced feeding easily results in vomiting. Frequently this forcing increases the resistance of the child. If the child is forced to swallow by filling the mouth with food or by closing the nose the irritation, fear and the feeling of suffocation causes renewed feelings of displeasure in the child which are soon associated with the process of food intake itself and which thus aggravate the resistance of the child against any kind of feeding. If children with anorexia are left hungry many of them seem to lack any feeling of hunger. The children appear content and after many hours or even days they still show no desire for food. Such prolonged hunger cures have a damaging effect on the nutritional state of the child. As suddenly as this anorexia appears it can also disappear again. At times the child can be brought to accept food by changing the person who feeds the child, with the feeding taking place preferably in the absence of the parents.

The therapy of anorexia must attempt to remove the disturbing memory which caused the refusal to eat. It is most advisable to give in to the wishes of the child. This offense against one of the most important basic rules of education will knowingly have to be borne for a time in order to maintain the child in an adequate nutritional state. Since the infant usually still accepts liquid food one contents oneself in giving the child only milk or milk mixtures and fresh fruit juices as vitamin carriers. By concentrating the food (condensed milk, addition of milk powder) and adding egg yolk the food intake is supplemented in quantity and quality. Such a procedure will still have to be chosen even in children at the end of the first year of life who then receive for a while only 800 to 1000cc milk per day. After two to three weeks during which no attempt must be made to give the child more solid food the memory of the unpleasant experience in feeding has usually disappeared. It is then possible to first re-introduce a cereal and later a fruit purée into the menu. But even this procedure can at times be unsuccessful. It then becomes necessary to find a substitute for the feeling of pleasure which normally accompanies the eating process. Swinging and singing, distracting the child with a toy and other procedures which are otherwise prohibited in the bringing up of infants at times become necessary in order to feed the infant and to prevent a further deterioration of the child's nutritional state. It is possible in this manner to combine the eating process with certain feelings of pleasure. In other instances seemingly very minor changes in the feeding technique may suffice to get the child to eat. A spoon of broth or fruit juice or a few drops of cold water between or with the individual spoons of food, a different cup or a new plate are at times sufficient to remove the resistance in older infants. In the most stubborn patients tube feeding at the rate of three to four times a day for several days or weeks might be the only solution. During this period no attempt should be made to feed the child with a bottle, a cup or a spoon.

The difficulties in food intake which develop during infancy not infrequently continue into the period of early childhood. Some of the children remain bad eaters for years. The feeding difficulties disappear only around the age of three to four years and part of the children then become good, even excellent eaters.

In the treatment of feeding difficulties no success can be expected from the use of drugs (hydrochloric acid, pepsin, vitamin B etc.). Gastric lavages do not cure anorexia. Change of the attending person is of advantage if in this manner the nervous environment can be completely eliminated and the care and feeding of the child be placed into the hands of a patient attendant. The last resort in treating these children with anorexia is their transfer to an institution where it is usually possible to help the children overcome this period of non-eating.

The influence of any febrile disease upon the nutritional processes is always more pronounced in neuropathic children than in normal children. Complete lack of appetite and refusal to accept even liquid food is frequent. Violent and repeated vomiting introduces the infectious disease and not infrequently persists throughout the entire period of temperature elevation. In the neuropathic patient the vomiting which frequently precedes the infection develops into so-called intractable vomiting. Treatment of this disturbance is not always simple. Interruption in food intake for 12 to 24 hours has been found most effective against this violent vomiting. During this period the child receives only 15% sugar water (glucose) in amounts of $^1/_2$ to 1 liter per day, at first in very small quantities (two to three teaspoons every 15 minutes) and later in larger portions. Following prolonged periods of vomiting the addition of a speck of salt to the sugar water is important in order to remove the frequently appearing hypochloremia. Some children prefer to take the liquid cool or even cold and in this form are less likely to regurgitate it. Some children like the addition of lemon juice to the liquid. Apart from this, sedatives bring about a central calming of the vomiting centers. At times it is also possible to obtain favorable results with a gastric lavage and subsequent filling of the stomach with an alkaline mineral water. If the lack of appetite persists in the nervous child beyond the first days of the infection an attempt will have to be made in all cases of longer lasting diseases to forcefully meet the nutritional requirements by concentrated feeding or even by tube feeding in order to avoid a deterioration of the nutritional state. For the treatment of anorexia which extends beyond the time of the infection hydrochloric acid and pepsin preparations have been found effective in some cases. In general an attempt will have to be made here too to bring about a weight gain in the child by infrequent, small, fatless meals until after some time the child again shows a readiness to accept food.

Diarrheas accompanying a parenteral infection, even in the neuropathic patient, require treatment only if the general condition is affected. The "irritability and weakness" of the intestines is so great in these children that the elimination of frequent, shreddy and usually mucous stools can develop already from minor causes. If all these diarrheas would be treated immediately with interruption or reduction in food intake the development of the child would come to a standstill within a short time. In these cases, too, only those forms of diarrhea require treatment which bring about disturbances in the general condition.

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This Book

Physiology and
Pathology of

INFANT NUTRITION

By L. F. MEYER, *and* ERICH NASSAU, M.D.

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